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VOL. II.—41ST YEAR SYDNEY, SATURDAY, JULY 10, 1954 No. 2

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Table of Contents.

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ORIGINAL ARTICLES— Page.

Nutrient Additives to Bread, by Eben H. Hipsley 45
Electrocardiographic Studies with Varying Potassium Plasma Levels, by E. R. Trethewie 51
Tracheo-Bronchial Cysts, with Report of an Unusual Case, by S. Livingstone Spencer and L. R. Finlay-Jones 56
The Intrabronchial Foreign Body, by A. G. McManis and Harry M. Windsor 57

REPORTS OF CASES—

Bronchogenic Cyst in Infancy, Causing Respiratory Obstruction, by Howard Williams 60
Basal Cell Carcinoma Occurring in Childhood, by K. Reye, M.B., B.S. 62
A Further Case of Acute Hæmorrhagic Leuco-encephalitis (Hurst), by R. V. Southcott and Malcolm Fowler 65

BOOKS RECEIVED 68

LEADING ARTICLES—

The Journal's Fortieth Birthday 69
Gentleness and the Approach to the Patient 69

CURRENT COMMENT—

An Experiment with Peptic Ulcer 70
The Prevention of Mental Illness 71
The Dietary Management of Peptic Ulcer and Ulcerative Colitis 71
Growth of the Hair 72
Pressure and the Bed-Sore Problem 72
Coronary Occlusion 73

ABSTRACTS FROM MEDICAL LITERATURE— Page.

Pathology 74
Morphology 74

SPECIAL ARTICLES FOR THE CLINICIAN—

CIV. The Teeth of Children 76

OUT OF THE PAST 77

CORRESPONDENCE—

The Training of Medical Students 78
Treatment of the Common Wart by X-ray Therapy 79
The Management of Chronic Hypoparathyroid Tetany 79

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA 79

PUBLIC HEALTH—

National Health Act, 1953: Pensioner Medical Service 80

AUSTRALIAN MEDICAL BOARD PROCEEDINGS—

Tasmania 80
Queensland 80

DEATHS 80

DIARY FOR THE MONTH 80

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 80

EDITORIAL NOTICES 80

NUTRIENT ADDITIVES TO BREAD.[1]

By EBEN H. HIPSLEY,

Nutrition Section, Australian Institute of Anatomy, Commonwealth Department of Health, Canberra.

In the preparation of modern white flour, which forms such an important item of the diet of many countries, substantial quantities of important vitamins and minerals are removed in the processing, and used in other than human diets. In many respects white flour is nutritionally inferior to wholemeal flour and to the so-called high extraction flour—that is, flour which preserves 75% or more by weight of the whole wheat grain. There are a number of reasons why white flour products are consumed in preference to wholemeal flour products, but it is not proposed to discuss these reasons here. The fact remains that large quantities of white flour are consumed.

Modern chemistry has made it possible to synthesize a number of vitamins and minerals at a comparatively low cost.

These two facts—that nutritionally inferior white bread is preferred, and that quantities of low-cost vitamins and minerals are available—has led to the practice in some countries (notably the United States of America, Canada and the United Kingdom) of replacing some of these substances which have been removed in the milling process. Furthermore, not only have the vitamins and minerals removed during the milling been restored, but in the case of some of these substances quantities have been added which more than restore the amount lost. In other words, flour (or bread) is being used as a convenient vehicle to raise the dietary intake of the presumably desirable substance. Thus in the United Kingdom since 1943, bread has been used as a vehicle for adding extra calcium to the diet; and in the United States of America since 1941, the riboflavin and calcium intake of the diet has been increased per medium of bread, whereas niacin, iron and thiamin have in both countries[1] been used in "replacement" quantities only.

Food Enrichment Policy of the United States of America.

The policy which governs the vitamin enrichment of foods in the United States of America has been defined in a set of principles which have been recommended by the Food and Nutrition Board. It is worth quoting here these

[1] Read at a meeting of the Australian and New Zealand Association for the Advancement of Science, Canberra, January 13 to 20, 1954.

[1] United States since 1941, United Kingdom since 1953.

principles, which were last revised and restated in April, 1953:

> (1) The Board, within carefully defined limitations, endorses the principle of the addition of specific nutrients to certain staple foods for the purpose of maintaining good nutrition as well as for correcting deficiencies in the diets of the general population or of significant segments of the population. The requirements for endorsement of the addition of a particular nutrient to a particular food include (a) clear indications of probable advantage from increased intake of the nutrient, (b) assurance that the food item concerned would be an effective vehicle of distribution for the nutrient to be added, and (c) evidence that such addition would not be prejudicial to the achievement of a diet good in other respects. These requirements have been met in the specific cases indicated in paragraph (6) below.
>
> (2) The Board emphasizes the desirability of meeting the nutritional needs of the people by the use of natural foods as far as practicable, and to that end encourages education in the proper choice and preparation of foods and the betterment of food production, processing, storage, and distribution so as to provide more fully the essential nutrients native thereto.
>
> (3) In order to avoid undue artificiality of food supply the Board favors, whenever practicable, the choice, as vehicles for the distribution of additional nutrients, of those foods which have suffered loss in refining or other processing, and recommends that the nutrients added to such foods should preferably be the kinds and quantities native to the class of foods involved.
>
> (4) The addition of other than natural levels of nutrients to foods which are suitable vehicles of distribution may be favoured when in the judgement of the Board the addition will be advantageous to the public health and when other methods for effecting the desired purpose appear to be less feasible.
>
> (5) Whenever technological and economic developments lead to extensive reduction in the consumption of a staple food, with a consequent nutritionally significant reduction in the intake of an essential nutrient or nutrients, the Board believes that consideration should be given to the desirability of restoring such nutrient or nutrients, to the dietary.
>
> (6) The Board reaffirms its endorsement of the enrichment of flour, bread, degerminated corn meal, and corn grits, the nutritive improvement of whole grain corn meal and of white rice, and the addition of vitamin D to milk, of vitamin A to table fat, and of iodine to table salt.

The present paper deals only with the question of the addition of nutrients to bread.

It is pertinent to examine the basis on which the decision was made in 1941 to add certain vitamins and minerals to American bread.

Although it was known that a few people in the United States of America died each year from pellagra and beriberi, at the time when the decision was made to enrich bread it could hardly have been held that frank beriberi and pellagra were national problems. Then, as now, reliable techniques designed to detect unequivocally the effects of lesser degrees of dietary deficiency were wanting. The facts were that when the results of the household dietary studies of Stiebeling, Monroe, Coons *et alii* (1941) were compared with a set of standards considered ideal by the National Research Council, it was inferred that many diets in the United States of America were unsatisfactory, and that there may have been a vast amount of vague ill health which could possibly be prevented by increasing the consumption of certain vitamins and minerals.

From such a (necessarily) ill-defined "base line", it is not possible to assess accurately the results of the bread enrichment policy. It is true that diseases such as beriberi and pellagra have become very rare in the United States of America since the introduction of bread enrichment; but as there have been concurrent changes in the diet pattern and in the economic situation of the people during recent years, the true value or otherwise of the bread enrichment programme is obscured.

A widely quoted piece of evidence in support of the United States of America policy of bread enrichment is that relating to Newfoundland. In 1944, a team of Canadian, American and British nutrition experts surveyed the nutriture and the diets of the St. John's and other areas of Newfoundland. It found a high prevalence of clinical signs and biochemical findings attributable to chronic deficiency of vitamin *A*, riboflavin and ascorbic acid. There was extensive dental caries, and although no pellagra was seen signs suggestive of chronic niacin deficiency of mild degree were encountered frequently. Peripheral neuritis was infrequent. Body weights showed evidence of being low, and many subjects had poor muscular development. Investigation of food supplies showed them to be below the National Research Council Recommended Allowance in thiamin by 32%, in riboflavin by 51%, in vitamin *A* by 69%, in ascorbic acid by 72%, and in calcium by 58%.

As a result of this survey it was recommended (i) that a nutrition education programme be undertaken, (ii) that a school milk scheme be instituted, (iii) that cod-liver oil and orange juice be distributed to vulnerable groups, (iv) that margarine be fortified with vitamin *A*, and (v) that flour be fortified with thiamin, niacin, riboflavin, iron and calcium.

As a result of a resurvey of Newfoundland in 1948 by the same team, the following conclusion was reached:

> Those signs and symptoms of malnutrition which could have been expected to decrease in prevalence as a result of the fortification of margarine with vitamin A and the enrichment of flour with riboflavin, niacin, and thiamin were less frequently encountered and less severe in 1948 than in 1944, whereas the prevalence of lesions which could not have been affected by these measures remained unaltered or actually increased.

In particular the team found no evidence that the ascorbic acid status of the groups surveyed, or of their diet, had improved.

From the evidence presented it is not possible for me to disagree with this interpretation of the reasons for the improvement noted, but the views of the authorities who made the report must be given due weight.

Bread Fortification Policy of the United Kingdom.

In 1941, before the introduction of the 85% extraction (national) flour, because of the difficulty of maintaining adequate thiamin intakes when white bread was used in the diet, and because synthetic thiamin could be made available reasonably cheaply, the British Government decided to enforce the addition of thiamin lost in the milling process. Before this policy could be fully implemented, owing to loss of shipping, stocks of wheat became dangerously low, and as an economy measure the extraction rate of flour was raised to 85%. As this flour was comparatively rich in thiamin, fortification became unnecessary. However, since it was thought that the phytic acid present in this flour might interfere with calcium absorption, and since the adequacy of the calcium intake of the diet was regarded as marginal, a decision was made to add calcium to the flour. Since the national flour was a comparatively good source of iron and niacin, it was not necessary to consider fortification of flour with these nutrients. The question of riboflavin fortification apparently was not considered—possibly partly because the riboflavin problem was solved by a national milk policy, and partly because suitable sources of synthetic riboflavin may not at that time have been available.

In December, 1952, however, a measure of "decontrol" of cereals was announced. The sale of bread made from white flour is now permitted, in the United Kingdom, but the bread is to be fortified by the addition of nutrients equivalent to the difference between 80% national flour, and the 70% flour. However, the superior quality of the 80% national flour receives official acknowledgement and encouragement from the fact that it is subsidized, whereas fortified 70% flour is not. The White Paper (1953) dealing with the matter states that "this [these measures] will ensure that freedom to produce whatever flour the public prefers will not result in any avoidable lowering of nutritional standards".

TABLE I.

Amino Acid Content of Various Types of Australian Bread Compared with Whole Egg Protein. (Calculated from Tables Published by Block and Mitchel, 1946–1947.)

Amino Acid.	Approximate Percentage Amino Acid Content of the Proteins of Milk and Bread.[1]						Percentage Deviation from Corresponding Value of Whole Egg Protein.			
	Milk.	White Bread.	Milk Bread (4% Added Skim Milk).	Wheatmeal Bread.	60/40 Wheatmeal Bread.	Whole Egg.	White Bread.	Milk Bread 4%.	Wheatmeal 100% Bread.	60/40 Wheatmeal Bread.
Arginine	4·3	3·9	3·9	4·2	4·1	6·4	−39	−39	−34	−36
Histidine	2·6	2·2	2·3	2·1	2·1	2·1	+5	+8	0	0
Lysine	7·5	2·0[2]	2·7[2]	2·7[2]	2·4[2]	7·2	−70[2]	−62[2]	−63[2]	−66[2]
Tyrosine	5·3	3·8	4·0	4·4	4·2	4·5	−15	+11	−2	−6
Tryptophane	1·6	1·0	1·0	1·2	1·1	1·5	−33	−33	−20	−26
Phenylalanine	5·7	5·5	5·5	5·7	5·6	6·3	−13	−13	−10	−11
Cystine	1·0[2]	1·9	1·7	1·8	1·8	2·4	−21	−28	−25	−25
Methionine	3·4[2]	1·0	1·3	2·5	1·9	4·1	−75	−68	−39	−53
Threonine	4·5	2·7	2·9	3·3	3·1	4·9	−45	−41	−33	−37
Leucine	11·3	7·5	7·9	6·8	7·1	9·2	−18	−14	−26	−23
Isoleucine	8·5	3·7	4·3	3·6	3·6	8·0	−54	−46	−55	−55
Valine	8·4	4·2	4·7	4·5	4·4	7·3	−42	−35	−38	−40
Glycine	2·3	7·2	6·5	—	—	2·2	+230	+200	—	—
Cystine-methionine	4·4	2·9	3·0	4·3	3·7	6·3	−55	−54	−34	−43

[1] Amino acid losses during baking have been ignored.

[2] Indicates limiting amino acids. Cystine and methionine are complementary.

Policies Regarding Nutrient Additives to Bread in Australia.

In Australia, the question of fortification of flour with thiamin received consideration in 1941 (Clements, Slater and Rial). Since Australian white bread was a better source of thiamin than white bread made in the United Kingdom and in the United States of America, and since there was no evidence of thiamin deficient diets on a national scale, thiamin fortification of bread was not recommended. Possibly because of the meat-eating habits of the Australian people, the question of iron and niacin fortification does not appear to have received serious consideration. A campaign to "drink more milk" had been launched, and this may have been one of the reasons why it was not thought necessary to consider riboflavin fortification of flour.

For several reasons it is considered that the time is now appropriate to reconsider the question of nutrient additives to Australian bread. Firstly, the cost of some of these nutrients has been reduced to a comparatively low level, and this fact diminishes the economic considerations which enter into the problem. Secondly, bread fortification practices have now been in operation in some countries for more than ten years, and it should be possible to make a more accurate assessment of their value. Thirdly, both the economic and the population structure of Australia has changed considerably in the past ten years, and this change may have affected the diet pattern.

In 1953, the National Health and Medical Research Council Advisory Committee on Food Additives reported as follows:

> The committee is unable to satisfy itself that there may not be in certain areas a significant amount of subclinical vitamin deficiency which would be effectually remedied by the addition of vitamins to bread . . . there are no valid reasons for prohibiting the addition of vitamins to flour and bread provided no misleading claims for the flour or bread are publicly made and provided the quantities added are effectually controlled.

It was further recommended that the "vitamin added" bread should contain the following substances in the proportions stated, in milligrammes per pound: thiamin 2·0 to 2·5, riboflavin 1·2 to 1·5, niacin 16·0 to 20, iron 13·0 to 16·5. No recommendation was made by this Committee with regard to calcium. In the following discussion I have assumed that "enriched" bread would have calcium added to bring it up to the levels recommended in the United Kingdom and the United States of America—that is, 500 to 625 milligrammes per pound.

Nutritive Value of Various Types of Bread.

At this stage of the discussion it is of interest to compare certain types of Australian bread in respect to certain specified nutrients.

Wholemeal bread, including 60/40 wholemeal bread,[1] is a slightly better source of protein than white bread, and the amino acid mixture is superior with respect to lysine and tryptophane (Table I). It has a great deal more thiamin, niacin and iron, and slightly more riboflavin and calcium than white bread.

Enriched white bread is equal to wholemeal bread in thiamin, niacin and iron, and is greatly superior in riboflavin and calcium; but like white bread (non-enriched), it is somewhat inferior to wholemeal with regard to the biological value of the protein.

It must be remembered that the nutrients which appear in the National Research Council (United States of America) Tables of Recommended Allowances occur in association with other nutrients known to be important in the metabolism, at least of animals. Restoration of the few nutrients listed in these tables will not restore the nutrients of the group with which they are usually—or invariably—associated. Although there is little evidence that the other nutrients are ever deficient in the diets of man—with the possible exception of folic acid and vitamin B_{12}—let us admit ignorance and recognize that time may tell a different story.

No one today denies that wholemeal bread is not nutritionally superior to white bread, whether the latter is enriched with vitamins and minerals or not. However, it is claimed by the proponents of enriched white bread that enriching enhances the value of white bread in the diet.

The Place of Bread in the Australian Diet.

The only up-to-date figures available relating to the national diet are those compiled in 1951–1952 by the Commonwealth Bureau of Census and Statistics relating to foods moving into civilian consumption from the wholesalers to the retailers. These figures show that 28% of the total Calories moving into civilian consumption were provided by cereal products (Figure I).

In a household food consumption survey carried out by the National Health and Medical Research Council in 1944, it was found that the per-capita consumption of bread was 7·2 ounces per day, and, whilst it is not entirely valid to assume that this is the daily bread component of the 28%

[1] "60/40 wholemeal" is one type of "wholemeal" bread commonly sold in Australia. It is made by mixing 60% wholemeal flour and 40% white flour.

of Calories, it is approximately true to infer that 14·5% of the total Calories of the diet are contributed by bread. By far the largest proportion (approximately 97%) of this bread is white bread.

Trends in the Australian Diet.

Statistics published annually by the Commonwealth Bureau of Census and Statistics, relating to foods moving into civilian consumption, accurately reflect trends in diet in Australia.

If the pre-war consumption (average, 1936–1937 and 1938–1939) of the main foods is compared with the consumption in 1951–1952 (Figure II), it is apparent that meat consumption has declined by 14%. This decline has been offset by a rise of milk consumption by 20% and of grain products by 8%. The decreased meat consumption has been partly responsible for a small decline in niacin (5%) and iron (2%), whilst the appreciable rise in calcium

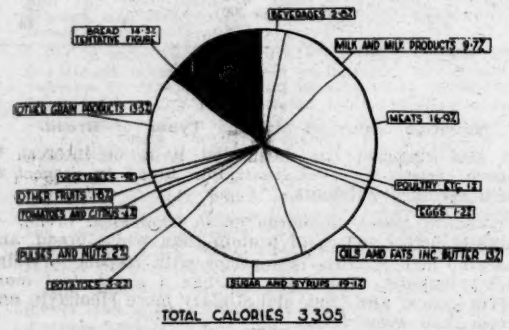

FIGURE I.

Sources of Calories in the Australian diet, 1952-1953. (From "Report on Food Production and the Consumption of Foodstuffs and Nutrients in Australia", Report Number 7, Commonwealth Bureau of Census and Statistics.)

(20%) and riboflavin (12%) has been due for the most part to the increased consumption of milk and milk products other than butter.

It is not suggested that the slight decline of niacin and iron indicated by these data is significant in itself, but it does indicate a trend which may be more pronounced in some sections of the Australian population. How far this is true, we can only surmise.

The Nutrient Composition of the Australian Diet.

The National Research Council Recommended Allowances[1] have been widely used as a yardstick for evaluating the dietary status of a population. It should be especially noted that it is not valid to say that because a group of individuals do not consume the recommended allowances of any or all of the nutrients, they are receiving insufficient quantities of food for optimal health, or suffering from malnutrition. How far the "average" Australian diet meets these recommended allowances[2] is shown in Figure III. It can be seen that all the recommended allowances are met with the exception of thiamin (93%), vitamin *A* (99%), and calcium (77%).[3]

Let us consider the actual contribution which various types of bread could make to the intake of specified nutrients in the Australian diet.

Thiamin.—The total thiamin content of the average Australian diet is slightly below (93%) the National Research Council Recommended Allowance. Approximately 15% of this thiamin is provided by bread. If 60/40 wholemeal bread[1] was to be used in place of white bread, 30% of the thiamin would be contributed by bread, and the recommended allowance would be more than adequately met. The thiamin pattern of a diet in which enriched bread is used is very similar to that in which 60/40 wholemeal bread is used (Figure IV).

Niacin and Iron.—Niacin and iron, which occur together in the main foods which make up the Australian diet, can be considered together. When white bread is used exclusively the "average" Australian diet does supply the recommended allowances of niacin and iron (Figure V). But, as has been previously indicated, this adequacy depends on the maintenance of the present levels of meat consumption. Both 60/40 wholemeal bread and enriched

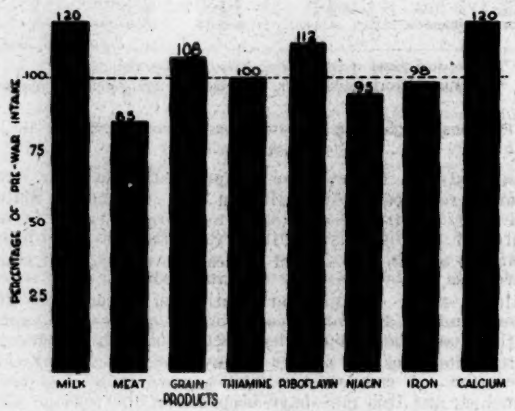

FIGURE II.

Selected foods and nutrients in the Australian diet. Intake per head, 1951-1952, as a percentage of average pre-war intake.

bread give a much wider safety margin of niacin and iron consumption (Figure V).

Riboflavin and Calcium.—Bread, whether white or wholemeal, is a poor source of riboflavin and calcium, and it makes a negligible contribution of these nutrients to the diet (riboflavin 0·1%, calcium 2·6%) (Figure VI). However, it has been recognized in the United States of America, in the United Kingdom and in Canada that bread is an efficient vehicle for adding extra nutrients to the diet. In the United States of America and in Canada riboflavin and calcium are added to bread in much greater quantities than would be present in wholemeal bread. Significant quantities of these nutrients (16% to 18%) are thus provided by enriched bread.

The Place of Bread in Different Types of Diets.

Obviously not all people consume exactly the same type of diet; for example, there are pure vegetarian diets, lacto-vegetarian diets (that is, those in which milk products are consumed in addition to vegetables), various therapeutic diets, and the diets of people who for economic reasons must curtail their expenditure on more expensive items such as meat, eggs and fruit.

Let us consider what happens to the nutrient intake when a person continues to eat white bread, but finds it necessary, for instance, to replace meat with cheese. The iron and niacin contents will fall to undesirably low levels (Figure III). Of such a lacto-vegetarian diet, it can be said that ill health may result unless substantial quantities

[1] In order to set goals for nutritional planning, the National Research Council (United States of America) examined the available evidence and compiled "Tables of Recommended Allowances" (at present under review). These tables have been widely used by many countries in the planning of diets.

[2] In this discussion we have compared the "average" Australian diet with the recommended allowances for a moderately active male.

[3] Many people consider that the recommended allowances for calcium have been set at too high a level by the National Research Council.

[1] See footnote, page 47.

of wholemeal[1] bread are also used (Figure III). Fortunately, most people who are lacto-vegetarians for religious reasons are aware of this fact; but unfortunately the need for wholemeal is sometimes not known by other people who adopt this type of diet.

It is often recommended that cheese can be used to replace all or part of the meat in the diet. However, it should be appreciated that if this recommendation is made, it should also be emphasized that more wholemeal bread should be eaten.

A common method used by people to economize in expenditure on food is to reduce the meat consumption and to increase isocalorically the consumption of bread. Thus if the consumption of meat was to be halved when white bread was used, the niacin intake would fall from 118% of the recommended allowance to 85%. With 60/40 wholemeal bread 98% of the recommended allowance of niacin would be met. A similar pattern is followed in the case of iron.

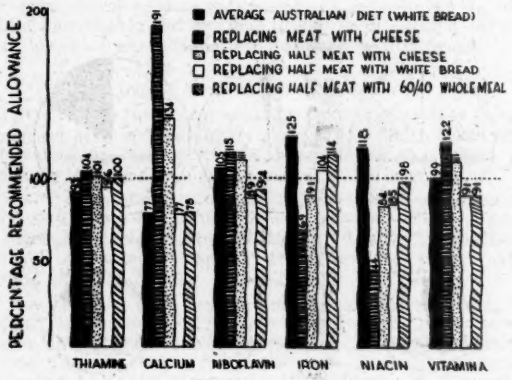

FIGURE III.
Percentages of recommended allowances met by various types of diet.

Nutritional Deficiency Diseases in Australia.

An examination of the demography reports[2] since the year 1914 reveals that up to about the year 1926 there was a substantial number of deaths from beriberi among males in Western Australia. Evidence is available to show that the majority of these cases occurred amongst Asiatics working on the pearling grounds. A few deaths from beriberi have been reported each year during recent years. It is probable that most of these cases would occur amongst Asiatics living on rice diets. Thus Maddox (1944) states that there were 20 patients suffering from beriberi admitted to the Royal Prince Alfred Hospital in the previous thirty years, all Chinese. During the same period two patients with pellagra were admitted.

From one to six deaths from pellagra each year have been reported over the past thirty years. A search of the literature covering a similar period revealed reports of 13 cases of pellagra, another five cases being mentioned for which no details are given except that they occurred amongst drovers working in the Northern Territory who had been living on diets of high carbohydrate, low protein and low vitamin contents.

The 13 cases mentioned above had the common feature that very little, if any, meat was eaten, and that the diet consisted largely of bread or puddings. In one case, a boy of six years had been living for some time on milk (one quart per day) and beef tea. In none of these cases was it reported that death occurred. It is probable that cases of pellagra occur which are not seen, or, if they are seen, are not reported in the literature, but the numbers cannot be very great. However, the fact that such cases of dietary deficiencies do occasionally occur raises the question whether there is any ill health in Australia due to deficiencies short of these frank and well-recognized diseases. Priestley (1952) has made the following statement:

> Deficiencies of these vitamins [niacin, thiamine and riboflavin] are much commoner than is generally suspected. Very many cases of chronic nervous exhaustion or of mild neurasthenia are due to deficiency of, mainly, thiamine, but commonly of other members of the group too. The administration of the whole complex, or at any rate of thiamine, riboflavin and niacin, often brings about a dramatic improvement in the patient's outlook and in general health.

De Garis (1950) described four cases in which neuritic symptoms were present which responded to treatment with preparations containing the vitamin *B* group. The diet pattern of none of her patients was described.

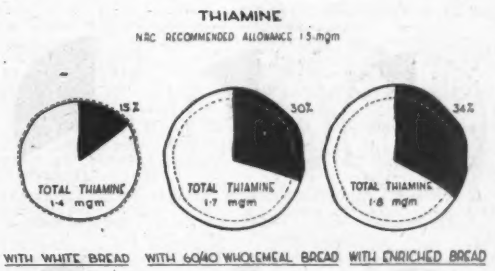

FIGURE IV.
Contribution of bread to total thiamine content of average Australian diet (*per capita* per day).

Clements (1942) produced evidence to show that in Canberra in 1942, in a series of 150 infants breast fed to the age of six months, 12 (8%) showed clinical symptoms characteristic of vitamin B_1 deficiency, which disappeared with increased vitamin B_1 intake. One inference which could be drawn from this report is that the thiamin intake of a significant proportion of women in 1942 was inadequate. This conclusion was perhaps given some support by the findings of the 1944 survey of household diets by the National Health and Medical Research Council, which found that in various States of Australia, from 20% to 66% of the households had unsatisfactory intakes of thiamine when judged by the National Research Council Recommended Allowances (Restricted).[1]

It is common knowledge that medical practitioners often prescribe vitamins for their patients, a fact which suggests that clinical (or subclinical) manifestations of vitamin deficiencies are not infrequent in Australia.

It would appear from the foregoing evidence that there is a doubt whether or not a substantial section of the population would benefit by the addition of thiamin, riboflavin, niacin, iron and calcium to white bread.

Present Practices Regarding Enrichment of White Bread in Australia.

In Australia, the National Health and Medical Research Council (1953) has suggested to State Governments that they should permit the addition of similar amounts of nutrients to those used in the United States of America at

[1] 60/40 wholemeal bread.

[2] Commonwealth Bureau of Census and Statistics.

[1] The "National Research Council Recommended Allowances (Restricted)" is a term adopted by the combined Food Board on *Food Consumption Levels in Australia and the United Kingdom*, 1944. The allowances are the original National Research Council figures for Calories and protein in all cases, and for minerals and vitamins in respect of groups up to the age of twenty years and expectant and nursing mothers. Elsewhere they are 70% of National Research Council recommendations for minerals and vitamins for adult men and women.

the present time. Although "enriched bread" has no legal authority in Australia, it is sold in some States apparently with the connivance of government authorities.

The enrichment of bread can be carried out either in the bakeries where especially prepared tablets (or wafers) containing the specified vitamins or minerals are dissolved in water and added to the dough mixture, or the nutrients can be added to the flour stream at the mill. The latter method, which has obvious advantages, has almost entirely superseded the former method.

Cost of Enriched Bread.

At the present time the cost of adding the nutrients to the flour is approximately £1 (Australian) per ton of flour. An increase of £1 per ton of flour would add one farthing to the retail cost of a two-pound loaf.

The cost to the Australian people would be 1s. 4d. per head per year, or a total of £570,000 for the present population.

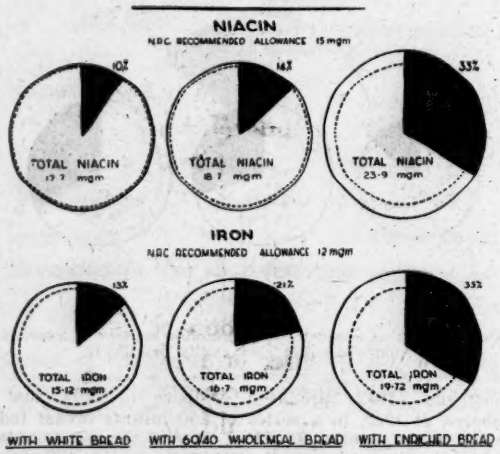

FIGURE V.

Contribution of bread to total niacin and iron content of average Australian diet (*per capita* per day). Interrupted circle represents National Research Council Recommended Allowance.

The vitamin-enriched bread available in some large cities at the present time is sold as a fancy bread with other additives such as gluten. It costs considerably more than the amount indicated by my calculation. At its present cost it is difficult to see how it could make any significant contribution to the diet of the Australian people—least of all to the diet of those who might benefit most.

The extra cost involved in bread enrichment is small (1s. 4d. per head per year), and it could easily be absorbed by the consumer. There is no conclusive evidence that the health of a significant number of the population would improve, but there is a distinct possibility that this would prove to be the case. There is no evidence that vitamin enrichment is, or is likely to be, harmful. In my opinion, those sections of the population which consume white bread should be given an opportunity to purchase enriched bread at reasonable cost and thus be in a position to reap any possible benefit.

Milk Bread.

Milk is added to bread for three main reasons: (i) to improve the baking quality of the bread (appearance, texture, flavour); (ii) to improve the keeping quality; (iii) to improve the nutritive value.

No specifications for milk bread are contained in any of the regulations of the pure food Acts of the various States of Australia. However, the New South Wales Department of Public Health and the Prices Commissioner have defined milk bread as that containing 4% added skim milk powder. In the United States of America, where most of the bread sold contains added milk, 3% is the average proportion of milk powder added.

Early experiments were carried out mainly with the idea of adding milk to bread for the purpose of improving its nutritive value. The addition of 6% dried skim milk to Australian bread was found by Hutchinson, West and Jeffreys (1947) to result in loaves which were immature, with poor volumes, harsh crusts, and coarse open crumbs. When the skim milk powder contained 20% fat, satisfactory loaves were obtained.

During recent years the Australian Bread Institute, in conjunction with the Commonwealth Scientific and Industrial Research Organization, has developed a special skim milk powder containing glyceryl monostearate in soluble form, which has produced loaves with at least the same volume as ordinary white bread, a softer texture, a higher

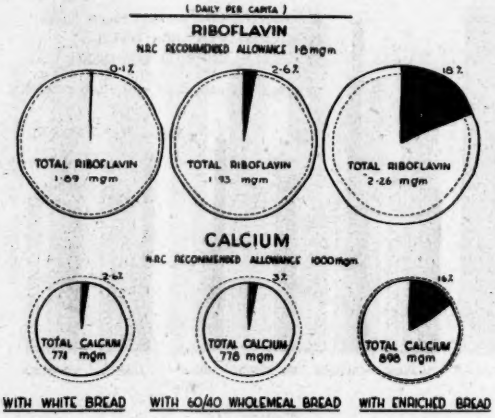

FIGURE VI.

Contribution of bread to total riboflavin and calcium of average Australian diet (*per capita* per day). Interrupted circle represents National Research Council Recommended Allowance.

crust colour, and improved keeping and toasting qualities. The general type of bread is, however, not very different from that which is at present sold in Australia.

What contribution to the diet would be made by the extra milk which would be consumed if milk bread (4% added skim milk) was to be the type of bread eaten to the exclusion of all other types?

The only nutrients which need be considered are protein, riboflavin and calcium.

The actual protein contributed by the milk of milk bread would be 0·8 gramme per day, which represents about 1% of the total protein content of the diet. The amounts of riboflavin (0·043 milligramme—2·3%) and calcium (30 milligrammes—3·8%) contributed to the total diet by the extra milk are likewise almost negligible. It is true that milk protein tends to supplement the protein of bread, as can be seen by referring to Table I, but at the 4% level of added skim milk this effect is not very great.

If milk bread was to be consumed exclusively, this would cost the average consumer £2 15s. extra per year. The extra milk[1] consumed in the bread would cost 14s. if purchased retail as whole milk. Even when the advantages of the automatic consumption of a mixture of cereal and milk protein are taken into consideration, it is obvious that milk bread is not an economical method of purchasing milk.

[1] Liquid whole milk contains fat and vitamin *A* in addition to the nutrients contained in the skim milk equivalent.

However, this does not mean to discount the advantages of milk bread with regard to baking, toasting and keeping qualities. It should be pointed out, too, that if milk bread was to be baked and handled in the ordinary fashion, so that the only additional cost would be that of the milk powder, milk bread would be an economical method of purchasing milk. At present this is not so.

Gluten Bread.

Gluten bread, which is sold under several trade names, has not yet been defined under regulations of the pure food Acts of the States. It enjoys a wide popularity because of its texture and its toasting and keeping qualities. It has been called "starch reduced bread", and at one time enjoyed a vogue in slimming diets. However, the term is really a misnomer. The larger volume results in the slices containing less solids (and hence fewer Calories) than slices of ordinary white bread; but weight for weight there are no differences. It has also been named on occasions "protein-enriched bread". Since the amount of protein added would make no significant contribution to the diet, the term "protein-enriched bread" is misleading to the public and should not be used. The term "gluten bread" is an accurate and adequate description of this bread.

Iodine Enrichment of Bread.

Goitre occurs in several areas of Australia, notably in the Gippsland area of Victoria, in parts of the South Coast and Tablelands of New South Wales, and in the whole of Tasmania.

In the Netherlands since 1943, bread has been successfully used as a vehicle for iodine in goitrous areas. Under a government regulation bakers of bread in specified goitrous areas are required to use iodized salt in the making of such bread.

I have carried out investigations in this method, and, as a result of a number of reports submitted to the National Health and Medical Research Council, a recommendation by the Council (1953) has been made to the States that bakers of bread in specified goitrous areas in Australia should be permitted to use iodized salt (one part of alkaline iodide in 10,000 parts of salt) in the baking of bread.

Summary and Conclusions.

The history of the policies regarding the addition of nutrients to bread in the United States, in the United Kingdom and in Australia is traced. It appears that it is an opportune time to reconsider the value of bread fortification.

The nutritional values of white bread, of 60/40 wholemeal bread, and of white bread "enriched" to the United States specification, are discussed; 60/40 wholemeal bread is superior to the other two types of bread.

The decreased post-war consumption of meat in Australia indicates that in some diets at least there will probably be a fall in the niacin and iron intake, unless more wholemeal bread is consumed, or unless these two nutrients are added to white bread.

It is evident that frank deficiency disease is uncommon in Australia, except amongst alcoholics and those people living on "unusual" diets. Although there is very little evidence available on which to base an opinion, it is possible that there may occur an appreciable amount of vague ill health which would be prevented by a higher intake of some nutrients.

Enriched bread is available in some of the larger capital cities of Australia, where it is sold as a special loaf at a premium price. Under these conditions it could not make any significant contribution to the health of the Australian people; but if it was sold at, or slightly above, the ordinary price of bread, it might make a significant contribution.

The demand for "milk" bread depends on its qualities of texture, toasting and keeping. From a nutritive point of view, the contribution of the added milk is insignificant.

The terms "starch reduced" and "protein enriched" bread are misleading and are inaccurate descriptions of the nutrient content of these loaves. In my opinion, they should not be used. The term "gluten" bread would be an adequate description.

Bread appears to be a suitable vehicle for increasing the use of iodized salt in goitrous areas.

Acknowledgements.

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References.

AKROYD, W. R., JOLLIFFE, N., LOWRY, O. H., MOORE, P. E., SEBRELL, W. H., SHANK, R. E., TISDALL, F. F., WILDER, R. M., and ZAMECNIK, P. C. (1949), "Medical Resurvey of Nutrition in Newfoundland, 1948", *Canad. M. A. J.*, 60: 329.

BOTTOMLEY, R. A. (1944), "Wheat, Flour and Bread, with Special Reference to Enriched Flour", M. J. AUSTRALIA, 1: 165.

BULLMORE, H. H. (1931), "A Case of Pellagra", M. J. AUSTRALIA, 1: 783.

CANTOR, S. J. (1927), "Pellagra: A Clinical Study with Notes on Some Cases", M. J. AUSTRALIA, 1: 713.

CLEMENTS, F. W., SLATER, E. C., and RIAL, E. J. (1941), "Australian Bread: Its Vitamin B_1 Content and Its Importance in the Australian Dietary", M. J. AUSTRALIA, 1: 634.

CLEMENTS, F. W. (1942), "The Symptoms of Partial B_1 Deficiency in Breast-Fed Infants", M. J. AUSTRALIA, 1: 12.

DE GARIS, M. C. (1950), "Vitamin B Deficiency in General Practice", M. J. AUSTRALIA, 1: 598.

GILMORE, H. (1950), "Report of a Case of Pellagra", M. J. AUSTRALIA, 1: 700.

GOLDSMITH, G. A., DARBY, W. J., STEINKAMP, R. C., BEAM, A. S., and McDEVITT, E. (1950), "Resurvey of Nutritional Status in Norris Point, Newfoundland", *J. Nutrition*, 40: 41.

HUTCHINSON, R. C., WEST, H. E., and JEFFREYS, J. T. (1947), "The Addition of Skim Milk Powder to Australian Bread", *J. C.S.I.R.*, 20: 483.

JOSKE, R. A., and TURNER, C. N. (1952), "Studies in Chronic Alcoholism: I. The Clinical Finding in 73 Cases of Chronic Alcoholism", M. J. AUSTRALIA, 1: 729.

LAWTON, F. B. (1929), "Pellagra", M. J. AUSTRALIA, 2: 133.

MADDOX, K. (1944), "Remarks Concerning Vitamin Deficiency in the Australian Adult", M. J. AUSTRALIA, 1: 81.

OSBORNE, T. B., and MENDEL, L. B. (1914), *J. Biol. Chem.*, 17: 325.

OSBORNE, T. B., and MENDEL, L. B. (1919), *J. Biol. Chem.*, 37: 557.

PRIESTLEY, H. (1952), "Vitamins in Medicine", M. J. AUSTRALIA, 2: 754.

SHERWOOD, J. E. (1949), "Pellagra", M. J. AUSTRALIA, 1: 390.

STIEBELING, H. K., MONROE, D., COONS, C. M., PHIPARD, E. F., ADELSON, S. F., and CLARK, F. (1941), "Family Food Consumption and Dietary Levels, Five Regions", U.S.A. Department of Agriculture, Miscellaneous Publication Number 452.

UNITED KINGDOM (1952), "Decontrol of Cereals and Feeding Stuffs", Her Majesty's Stationery Office.

WETTENHALL, R. R. (1925), "Pellagra", M. J. AUSTRALIA, 2: 59.

ELECTROCARDIOGRAPHIC STUDIES WITH VARYING POTASSIUM PLASMA LEVELS.[1]

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WHILE the changes in the electrocardiogram associated with varying potassium levels in the plasma are regarded clinically as well known, it was considered desirable to determine the changes in the electrocardiogram when the leads A, B, C (Trethewie, 1951, 1953) were employed. This triple display is regarded as more complete and valuable clinically than the usual display I, II, III, aVR, aVL, aVF, V_1 to V_6.

Further, it became apparent that the changes usually attributed to potassium level changes may not be due to the level of potassium, but to secondary effects which we

[1] This work was aided by a grant from the National Health and Medical Research Council.

were able to analyse because of the completeness of the blood analysis carried out on these patients by Dr. McDonald and Dr. Coats.[1] This feature has been pointed out by Merrill, Levine, Somerville and Smith (1950).[2]

Again, we have carried out investigations on the isolated perfused heart in which potassium levels alone can be varied, and have recorded the findings corresponding to lead C (termed "lead C equivalent").

Method.

Patients.

Electrocardiographic examinations were made on patients with hypopotassæmia and hyperpotassæmia with "General Electric" and "Cardiotrace" machines. Lead A (Trethewie, 1951) was led from the *manubrium sterni* and the xiphisternum, lead B from the mid-axillary line (at the horizontal level of the xiphisternum) and the xiphisternum, and lead C from the back to the right of the mid-line (at the horizontal level of the xiphisternum) and the xiphisternum, the angle between leads B and C being a right angle. The xiphisternum lead corresponds to the left arm lead in lead I (Einthoven). All three leads are at right angles to each other with a common origin (the xiphisternum).

Heart Perfusion.

Cats were anæsthetized with ether, and 0·7 millilitre of heparin (1000 units per millilitre) was injected intravenously. The heart was perfused via the aorta with a solution (through which varying proportions of 5% and 10% carbon dioxide in oxygen was bubbled) containing in flask A (Figure I) sodium bicarbonate 2·99 grammes, potassium chloride 0·8 gramme, sodium chloride 14·42 grammes, anhydrous sodium acid phosphate 0·21 gramme, calcium chloride 0·2 gramme, magnesium chloride 0·16 gramme, and dextrose 2·0 grammes in two litres. Flask B (Figure I) contained the same solution except for the potassium chloride concentration, which in the "low potassium" series was 0·32 gramme and in the "high potassium" series was 1·56 grammes per two litres. This meant that the potassium level varied from two to 10 milliequivalents per litre, while the chloride ratio was altered very little. The pH of the perfusing solution was estimated throughout the perfusion, and samples were taken for analysis of potassium (photometrically), carbonate and other ions. The temperature of the delivered perfusion fluid was recorded regularly (maintained at approximately 37° C.) and also that of the bath in which the heart was suspended (Figure I). Lead "C-equivalent" for the electrocardiogram was taken from the front to the back of the heart at the margins of the bath at the level of the heart apex (Figure I). The recording was made on a cathode ray oscillograph and photographed on 35 millimetre film.

Experimental Investigation.

The isolated heart of the cat was used and perfused as described.

Low Potassium Level.

In Figure II we note that the electrocardiogram taken (i) with a normal potassium level (4·3 milliequivalents per litre) is approximately normal (lead "C equivalent"). A steep *P* wave is noted in association with empty auricles. Half a minute after the solution containing potassium 2·5 milliequivalents per litre had been issuing from the heart (after flask A had been clipped off, the clip to flask B opened, and washing out effected by the opening of clip C—Figure I), the electrocardiogram (ii) was unchanged, and it was still unchanged after one minute (iii). Three minutes after the alteration a sag after the *S* wave appeared (iv), which increased at nine minutes (v) and eleven minutes (vi). Between (v) and (vi) the solution in the bath in which the heart was suspended (Figure I) was changed completely to a similar one, but containing 2·5 milliequivalents of potassium per litre, with no alteration in the electrocardiogram. This was also true of a normal electrocardiogram when only the solution around the heart (not perfused) was changed.

When the potassium level is reduced sufficiently the heart goes into complete heart block after a delay in the conduction time. This is shown in Figure III, in which tracing (i) shows the *P-R* time to occupy 0·45 second. The potassium level had been reduced to 2·8 milliequivalents

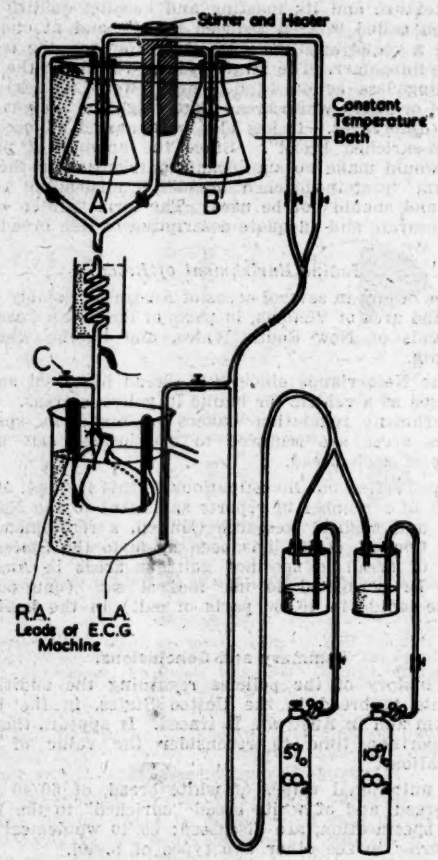

FIGURE I.

Diagram of apparatus used in perfusion of cat heart. Details in text. (Diagram by courtesy of Mr. K. Shankly.)

per litre six minutes previously. One hour after the potassium level had been reduced the first time, it was further reduced to 1·6 milliequivalents per litre, and twenty-three minutes later a tracing (ii) showed that the heart had passed into complete heart block. This electrocardiogram originally showed elevation of the *S-T* segment, which became reduced to the isoelectric line when the potassium level was reduced. It is noticed (ii) that the *T* wave is still high, even when the potassium level is 1·6 milliequivalents per litre.

High Potassium Level.

In Figure IV we note that the electrocardiogram (lead C) taken (i) with normal potassium level (4·3 milliequivalents per litre) showed an alternating height to the *T* waves, but was otherwise normal. Half a minute after the potassium issuing from the heart was increased (to

[1] Acknowledgement is gratefully made to Dr. I. R. McDonald and Dr. D. A. Coats for allowing the fullest use of their findings in patients with anuria.

[2] More recently, Roberts, Melville and Magida (1953) have presented evidence of "hyperpotassæmic" findings produced by other substances.

8·5 milliequivalents per litre) the electrocardiogram (ii) was normal. Two and a half minutes later a biphasic wave form with absent *P* developed (iii). Three and a half minutes after the potassium issuing from the heart was increased the tracing became irregular (iv), and it was

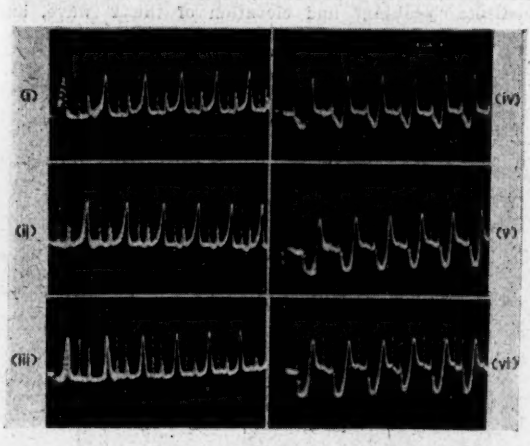

FIGURE II.

Electrocardiogram lead C "equivalent" of perfused cat heart, showing development of sag, but little change in *T* wave with potassium reduced to 2·5 milliequivalents per litre. Ordinate marker: 1·0 millivolt. Time: 0·2 second.

even worse at four minutes. Then the potassium level was changed to normal, and fifteen seconds later the electrocardiogram showed larger wave forms (vi), which

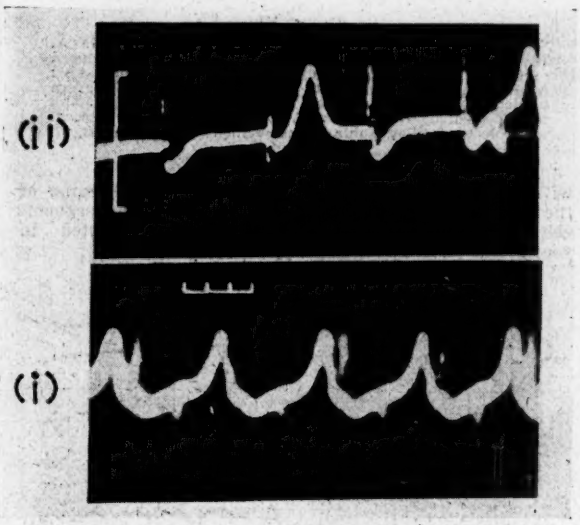

FIGURE III.

Heart block produced in heart perfused with low potassium solution, *T* wave still considerable. Further details in text.

became coupled at one minute (vii). At eleven minutes (viii) the *P* wave reappeared in a fairly normal trace.

Electrocardiographic Findings in Patients.

Hypopotassæmia.

The significant points in the history of one patient were as follows:

A., aged twenty-eight years, was admitted to hospital on July 29, 1951, with a history that for two days after the taking of a purgative vaginal bleeding had been present, the patient then being about nine weeks pregnant. Curettage was performed on her admission to hospital, in view of the diagnosis of *Clostridium welchii* septicæmia. Anuria developed at that time, and four days later the urinary output was still low (40 millilitres daily). The blood pH fell to 7·25 and the patient was drowsy. By August 21, the blood potassium level had fallen to 2·4 milliequivalents per litre, the pH was 7·18, and the bicarbonate level was 19 milliequivalents per litre. An electrocardiogram (Figure V) on the following day showed an inverted *T* wave in lead C, left axis deviation in lead B, a low take-off from the *S* to a biphasic *T* wave in lead A, and a *Q-T* interval of 0·26 second.

In the usual electrocardiogram (Figure V), there is a doubtful "low take-off" in lead II, and a biphasic *T* wave in lead V_2.

The patient was cyanosed on this day.

One litre of a potassium chloride solution (50 milliequivalents) with added vitamin B_1 was thereafter given intravenously.

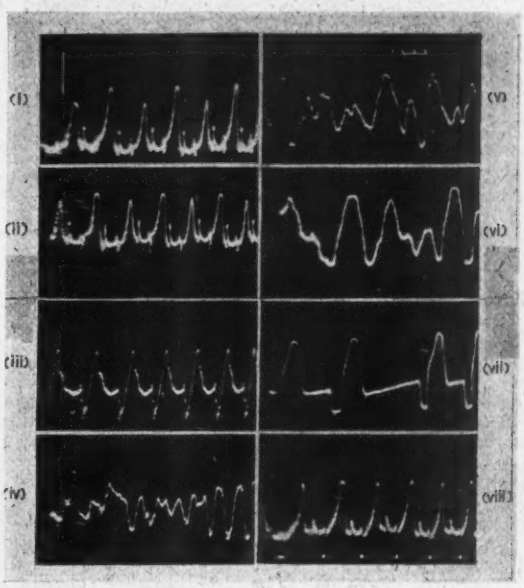

FIGURE IV.

Electrocardiogram lead C "equivalent" of perfused heart with high potassium level perfusion. Details in text.

On August 23 the patient was very cyanosed. The potassium level of the blood was 2·5 milliequivalents per litre, the pH was 7·35, and the bicarbonate level was 21·4 milliequivalents per litre.

The electrocardiographic findings (Figure V) were more pronounced: in lead C, inversion of the *T* wave was more evident; in lead B, inversion of the *T* wave had increased; in lead A, the low take-off from *S* had increased and the *T* wave was now reduced to a flat nearly isoelectric level.

On August 24 there was some subjective improvement, but the inversion of the *T* wave was greater in leads B and C (Figure VI); 50 milliequivalents of potassium were given intravenously on August 25. On that same day the electrocardiogram indicated considerable improvement. The patient's condition appeared comparable with that on August 22, with a slightly inverted *T* wave in lead C, a moderately inverted *T* wave in lead B, and a biphasic to upright *T* wave in lead A (Figure VI). The usual leads showed (Figure VI) a low "take-off" in leads II and V_6 with a biphasic *T* wave in lead II.

Potassium therapy was then discontinued. On August 26 the electrocardiogram again showed increased inversion of the *T* wave (Figure VII).

Potassium was again given (orally) with improvement on August 27, the electrocardiogram showing reduced inversion of the *T* wave (Figure VII). The full display (Figure VIII) in leads I, II, III, aVR, aVL, aVF and V_1 to V_6 was practically normal. The patient was allowed to sit out of bed on August 31.

On September 3 a nearly normal electrocardiogram was obtained (Figure IX), although the *T* wave in lead C was of small voltage (though upright). The inverted *T* wave in lead B is normal in the majority of subjects. Since the electrocardiogram had been taken previously with the patient lying down (owing to the severity of her illness), it was again taken with her lying down (Figure IX*b*) as well as sitting up (Figure IX*a*), the usual technique being adopted. The tracing was practically unchanged.

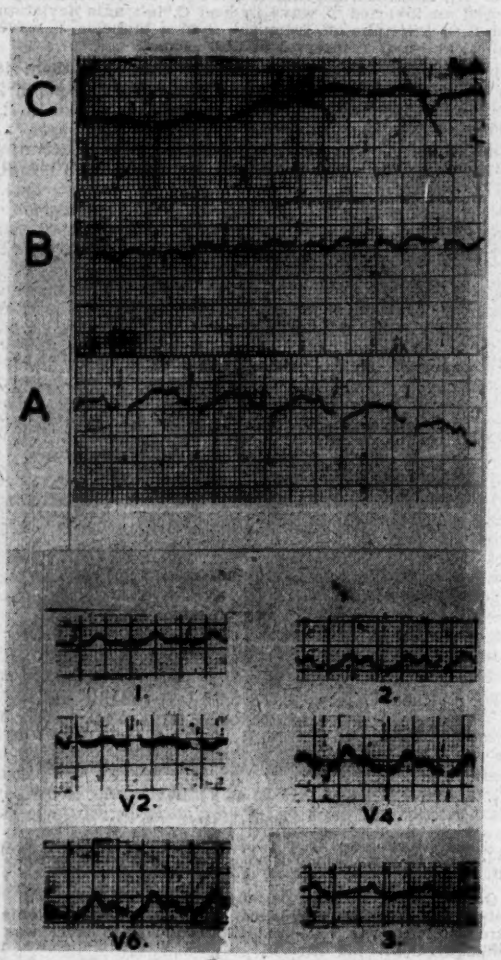

FIGURE V.

Electrocardiogram of A, leads A, B, C, showing signs of hypopotassæmia. Also leads I, II, III, V_2, V_4 and V_6.

After leaving hospital the patient again felt weak, and on February 14, 1952, it was noted that the *T* wave in lead C was again slightly inverted.

It is noteworthy that while it was evident with leads A, B and C that there was a return to the increased abnormality in the electrocardiogram after potassium therapy had been stopped on August 25, 1951, with return to a more normal tracing when it was recommenced, this change was not so evident with the ordinary leads.

It is also of significance that while there was pronounced subjective improvement on August 24, 1951, the electrocardiogram (both techniques) indicated a worsening of the abnormality. This aspect is important in the management of patients with potassium level changes in the blood. I have already presented evidence from animal experiments that not all the findings commonly attributed to hypopotassæmia are in effect due to the reduced potassium level alone.

Hyperpotassæmia.

The changes in the electrocardiogram of patients with an elevated serum potassium level are well known. These constitute "peaking" and elevation of the *T* wave, and

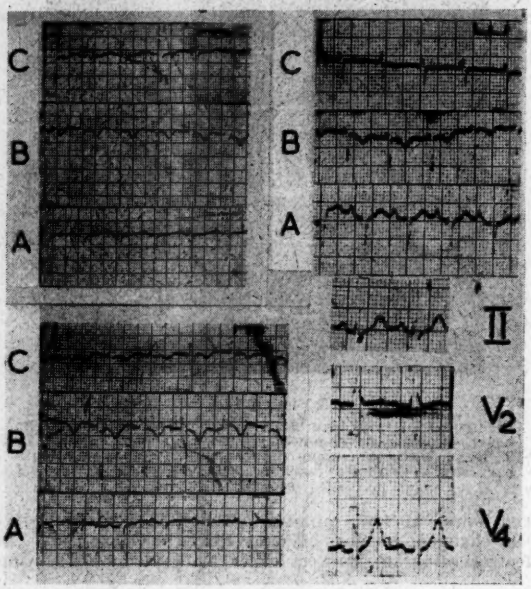

FIGURE VI.

Top left hand panel: electrocardiogram of A, leads A, B, C, one day after Figure V, showing more pronounced evidence of "hypopotassæmia". Bottom left hand panel: subjective improvement of patient A, but more severe evidence of "hypopotassæmia", leads A, B, C. Right hand panel: electrocardiogram of A, one day later, with clinical and electrocardiographic "improvement", leads A, B, C, II, V_2 and V_4.

with higher levels loss of the *P* wave and broadening of the *QRS* complex with a big biphasic swing and segment elevation. Finally, irregularity like that noted in ventricular fibrillation may appear before death.

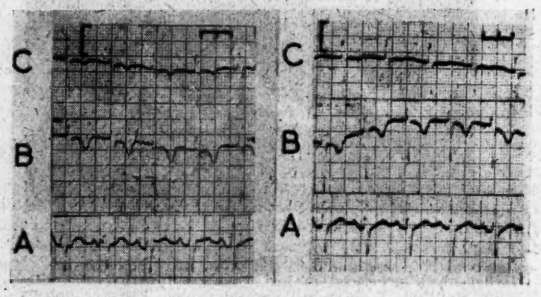

FIGURE VII.

Electrocardiogram of A (leads A, B, C), showing worsening following discontinuance of potassium therapy (left hand panel) and improvement in electrocardiogram one day later following recommencement of potassium therapy, leads A, B, C (right hand panel).

The significant findings in one case were as follows:

B., aged twenty-two years, was admitted to hospital on October 29, 1952, suffering from abortion with *Clostridium welchii* infection and anuria. A tracing on November 6 (leads A, B and C) showed a "peaking" of the *T* wave in

lead A and left axis deviation (large *S* wave in leads B and C) (Figure X). The patient had hypertension at this time. The plasma potassium level was then 7·1 milliequivalents per litre.

On November 10 the patient became worse, with impaired memory, and was passing only 150 to 180 millilitres of urine in the twenty-four hours. An electrocardiogram (full display) showed a big biphasic swing with absent *P* waves. Segment elevation was evident, maximal in lead V_2 (Figure XI). The tracing taken at the same time of leads A, B and

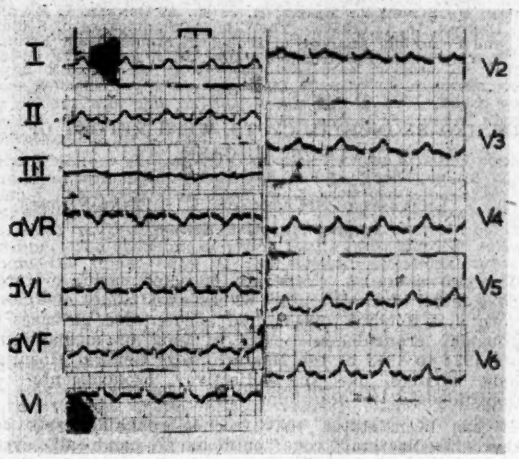

FIGURE VIII.

Patient A. Concurrent electrocardiogram to Figure VII (right hand panel), full display.

C (Figure XI) showed absent *P* waves, a big biphasic swing especially in leads A and C, with considerable segment elevation, and also an upright *T* wave in lead B (0·8 millivolt). The blood potassium level was 8·8 milliequivalents per litre.

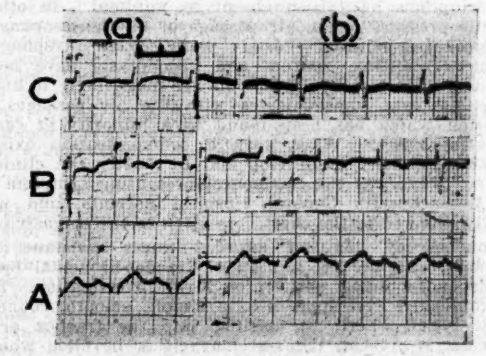

FIGURE IX.

(*a*) Electrocardiogram of A, leads A, B, C, patient sitting up. (*b*) Electrocardiogram of A, leads A, B, C, three days after clinical recovery, patient lying down.

Glucose (30 grammes) and insulin (20 units) were given intravenously, and twenty minutes later the electrocardiogram (full display) showed a reappearing *P* wave in leads I, II, V_1 and V_2 (Figure XII). The biphasic swing had largely disappeared. The *T* wave was peaked. However, the blood potassium level had fallen to only 8·4 milliequivalents per litre. The limit of sensitivity of the method employed for two samples tested together (as was done with these samples) is ±2%. The A, B, C tracing showed (Figure XII) an evident *P* wave in leads A and C, a peaked *T* wave in lead A (1·1 millivolts) and a flat *T* wave in lead B. The biphasic swing was no longer apparent.

Discussion.

Hypopotassæmia.

With leads A, B and C, the main features associated with hypopotassæmia are depression and inversion of the *T* wave, most pronounced in lead C, and a "low take-off" most evident in lead A. Lengthening of the Q-T interval

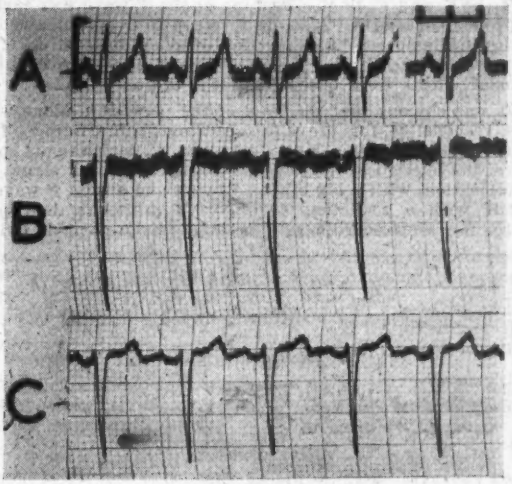

FIGURE X.

Electrocardiogram of B, leads A, B, C, showing peaking of *T* wave in lead A, in hyperpotassæmia.

is also found. Nothing further is learnt about the clinical condition by leads I, II, III, aVR, aVL, aVF, and V_1 to V_6, and in fact in the case cited changes in leads A, B and C were found to be a more sensitive index. Changes in the *T* wave in lead C were found to be the most sensitive index.

While we ordinarily recognize inverted and flat *T* waves, a low take-off, and a lengthened Q-T time as the hallmarks of a low blood potassium level, it is evident that in our

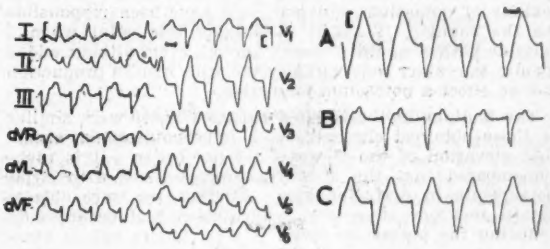

FIGURE XI.

Left hand panel: electrocardiogram of B, full display, showing gross evidence of potassium "intoxication" with absent *P* wave, segment elevation *et cetera* three days after Figure X. Right hand panel: concurrent electrocardiogram, leads A, B, C, patient B. Equally dramatic changes shown in the three lead display.

perfusion experiment with the potassium level reduced to figures as low as and lower than clinical levels, the *T* wave is never inverted or flattened. It is reduced in height, but not to any degree when allowance is made for the "sag". Some features distinguishing our experiment from the clinical picture are that we maintain our pH and our bicarbonate, chloride and sodium levels with newly delivered fluid. This might indicate that it is not the potassium level *per se* which flattens the *T* wave, but a secondary effect. It is known that if the potassium level is altered in the intact animal, concurrently the pH changes and an ionic shift occurs. This aspect will be explored in a later

paper. In the patient's electrocardiogram the *T* wave in lead C disappeared, while with much greater experimental reduction of the potassium level the *T* wave was still little altered in height.

Hyperpotassæmia.

We were able to record equivalent findings in leads A, B and C, and with the full display leads I, II, III, aVR, aVL, aVF, and V_2 to V_6. Peaking of the *T* wave was early, especially in lead A. Also, on a return to hyperpotassæmia following temporary amelioration of symptoms, an upright *T* wave in lead B (not recorded) appeared before definite "toxic" effects were observed in the ordinary leads.

It was obvious that the level of the potassium alone was not responsible for the electrocardiographic findings, for when the potassium level was 8·8 milliequivalents per litre the electrocardiogram showed pronounced signs of intoxication, with a big biphasic swing and absent *P* waves, while with a reduction of only 0·4 milliequivalent per litre, the electrocardiogram became fairly normal (with

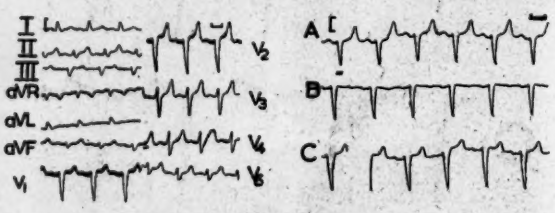

FIGURE XII.

Left hand panel: full display electrocardiogram, patient B, twenty minutes after glucose and insulin given intravenously immediately after Figure XI was recorded. Right hand panel: concurrent A, B, C electrocardiogram of patient B, indicating identical improvement.

both techniques). In the interval glucose and insulin had been given intravenously; this forces an intracellular migration of potassium. The fall is real, as the photometric readings for the high level were 61·0, 61·0 and 63·0, and for the low level 58·3 and 58·1 estimated at the same time. It may well be that the electrocardiographic findings were related to migration of ions across the membrane, rather than the level of potassium. However, it is to be remembered that glucose itself will reduce the toxicity of potassium and may well have been responsible for the change. We will investigate this point when a suitable patient again presents, though even without added insulin the intact body will respond with insulin production and so effect a potassium migration.

The findings in the isolated perfused heart were similar to those obtained clinically with hyperpotassæmia, except that elevation of the *T* wave did not occur. It is to be remembered that the *T* wave changes little with hypopotassæmia in perfusion also. Further, we were able to obtain recovery after initial fibrillation had occurred by reducing the potassium level.

Summary.

1. The findings in the A, B, C electrocardiographic display are recorded in hypopotassæmia and hyperpotassæmia in anuria.

2. No further information is obtained with the full display of leads I, II, III, aVR, aVL, aVF and V_2 to V_6, and in fact these tracings were not quite so sensitive in indicating a change as the A, B, C display in one patient.

3. The findings in the electrocardiogram with the isolated perfused cat heart are recorded when only the potassium level was varied.

4. The possible significance of these findings is discussed.

Acknowledgement.

Grateful thanks are tendered to Jacqueline Bladin and Margaret Hodgkinson for technical assistance with the heart perfusion.

References.

MERRILL, J. P., LEVINE, H. D., SOMERVILLE, W., and SMITH, S. (1950), "Clinical Recognition and Treatment of Acute Potassium Intoxication", *Ann. Int. Med.*, 33: 797.

ROBERTS, K., MELVILLE, M. D., and MAGIDA, M. G. (1953), "Electrocardiographic Alterations Produced by a Decrease in Plasma pH, Bicarbonate and Sodium as Compared with Those Produced by an Increase in Potassium", *Circulation Res.*, 1: 206.

TRETHEWIE, E. R. (1951), M. J. AUSTRALIA, 2: 718.

TRETHEWIE, E. R. (1953), "Simplified Electrocardiography", Department of Physiology, University of Melbourne.

WYNN, V., SIMON, S., MORRIS, R. J. H., MCDONALD, I. R., and DENTON, D. A. (1950), "The Clinical Significance of Sodium and Potassium Analyses of Biological Fluids: Their Estimation by Flame Spectrophotometry", M. J. AUSTRALIA, 1: 821.

TRACHEO-BRONCHIAL CYSTS, WITH REPORT OF AN UNUSUAL CASE.

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TRACHEO-BRONCHIAL CYSTS are of some rarity. Hardy (1949) found in the literature 82 cases and added one more. Brown and Robbins (1944) had encountered 21 cases of "probable" bronchiogenic cysts of lung or mediastinum from 1934 to 1943 at the Massachusetts General Hospital. (Some writers use the adjective "bronchogenic" or "bronchiogenic" in describing these cysts.)

It has been stated that tracheo-bronchial cysts are either "mediastinal" or "pulmonary", and all cysts, accounts of which have been traced by the present authors, have been situated within the mediastinum or lungs. The case now reported is of particular interest as the cyst lay in the neck, and further, since it had an unusual histological appearance.

Clinical and Surgical Features.

All but three of Hardy's series of 83 patients were over the age of eighteen years. The remaining three were infants. In a number of reported instances the discovery of the cyst has been incidental to an X-ray examination, and symptoms have been absent or minimal. In other patients pressure on an air passage or on the œsophagus has produced clinical effects. Pain, cough, dysphagia, dyspnœa, vomiting, sputum and hæmoptysis have been recorded. The last two symptoms are most likely to arise where the cyst communicates with the air passages, a condition which has been found in a minority of cases (Conklin, 1950). When such a communication exists, infection is likely to develop and to modify the clinical picture. Pain was the commonest presenting symptom in the Massachusetts General Hospital patients, and was usually referred to the chest, less often to the epigastrium.

Bronchoscopy has not afforded much assistance in diagnosis, and the recognition of tracheo-bronchial cysts has depended almost entirely on X-ray investigation. The uncomplicated cyst appears as a simple, smooth, rounded opacity; but where an opening into the trachea or a bronchus is present, the cyst may either be filled wholly with air, or may be partly filled with air and show a fluid level. Under these circumstances a diagnosis may be made of lung abscess.

When the cyst is small and not causing symptoms, it may be wisest to avoid surgical intervention and to follow the patient's progress by repeated clinical and radiographic examinations. On the other hand, when the cyst is distressing the patient, its removal must be contemplated, and in most cases thoracotomy will be needed. Complete removal is preferable to drainage, marsupialization or incomplete removal.

Sweet (1950) states that the technical difficulty varies with the location of the cyst, with the presence or absence of a communication with the air passages, and with the question of whether or not infection has become estab-

lished. Some cysts have been found to lie in the substance of the tracheal or bronchial wall, and Sweet emphasizes that in such cysts every endeavour must be made to preserve the integrity of the mucosa. When a communication occurs it is usually small, but the opening, or any perforation resulting from the removal of an intramural cyst, should be covered by a flap of pleura or by suturing the margins to the lung or the œsophagus.

Report of a Case.

A, a female office worker, aged twenty-two years, was referred to one of us (S.L.S.) by Dr. Colin Ratcliff. The patient complained of a swelling in the neck, present for six months, which she thought had been increasing in size. Slight difficulty in swallowing had been noticed for some years, but there was no complaint of cough or dyspnœa, and the patient was able to sleep lying flat in bed. There was also a history of an emotional upset, and of the loss of about twelve pounds in weight.

Examination showed the patient to be a slim young woman of composed demeanour and in good general condition. There was a rounded swelling of cystic consistency and with a diameter of about two or three centimetres in the lower portion of the neck, a little to the left of the mid-line. The swelling, of which the inferior third or so lay behind the upper edge of the *manubrium sterni*, moved readily on deglutition, and appeared to be contained in the lower part of the left thyroid lobe. The remainder of the thyroid gland was palpable and was slightly and diffusely enlarged. The pulse rate was 72 per minute and there was no tremor. X-ray examination of the chest revealed no abnormality.

A diagnosis was made of slight generalized colloid change in the thyroid gland, with a solitary adenoma and no evidence of toxicity. Removal of the "adenoma" was advised.

At Sydney Hospital on June 16, 1953, the patient's neck was explored through a conventional collar incision. Examination of the thyroid gland revealed generalized colloid enlargement, but it was found that the "adenoma" was in fact a cyst, with semi-opaque bluish-white walls, lying outside the fascial sheath of the thyroid gland, and below and slightly in front of its left lower pole. Apart from areolar tissue and a small vascular pedicle which disappeared backwards on the left side of the trachea, no obvious attachment to surrounding structures could be made out. The cyst was readily removed, but its exact nature was not recognized at this stage. As the thyroid was only slightly enlarged, none of the gland was removed, the wound being closed without drainage. (In view of the unusual histological features to be described below, it may be appropriate to emphasize that the cyst was surrounded by loose areolar tissue, which permitted its easy removal by finger dissection. It is felt that any suggestion that portion of one of the adjacent neck muscles adhered to the cyst wall and was removed with it may be rejected.)

The patient made an uninterrupted recovery, and when examined a month after the operation she was well and free from symptoms.

Pathological and Embryological Aspects.

The cyst measured 4·0 by 2·5 by 1·5 centimetres and had a smooth, slightly lobulated external surface. It was filled with semi-fluid greyish mucoid material. The lining was smooth and the cyst wall measured 0·1 centimetre in average thickness. Microscopic examination showed a lining of pseudo-stratified ciliated columnar epithelium with interspersed goblet cells. Lymphocytes were scattered through the underlying fibrous connective tissue. Small groups of mucous and serous glands were also found together with some hyaline cartilage. In one situation there were a few strands of voluntary muscle. Except for the last tissue, the structures described are those found in the trachea and bronchi (Figures I, II, III and IV). It may be significant that in sections of the cyst wall smooth muscle fibres were not found, although they have been described in many of the tracheo-bronchial cysts found in mediastinal or pulmonary situations.

Comment.

According to Laipply (1945) these cysts may arise in one of the following ways: (i) a pinching off of a diverticulum of the foregut in the region of the tracheal bud; (ii) a secondary budding of the tracheal bud itself; (iii) an abnormal division of the tracheo-bronchial tree at a later stage of development.

The trachea and bronchi arise from the ventral aspect of the foregut. Two longitudinal grooves develop and extend deeply towards the mid-line until separation of the trachea and œsophagus is complete. At this stage it is possible that a small bud of tissue could be cut off and pass caudally with the respiratory tract primordia. Tissue organizers may be potent enough to induce formation of an extra lung bud, but not powerful enough to cause the formation of properly organized lung tissue (Hirschfeld, 1951).

The cyst described in this paper showed two unusual features. Firstly, it lay in the neck rather than within the chest; secondly, its wall contained some fibres of voluntary muscle. It is suggested that these two features are related, and that they indicate an origin from cells lying near the junction of the main tracheal bud with the foregut.

Summary.

1. A brief account is given of tracheo-bronchial cysts.

2. A case is reported of a tracheo-bronchial cyst occurring in the neck and also having an unusual histological appearance.

3. The possible embryological basis is discussed.

Acknowledgement.

The photomicrographs were prepared by Mr. R. A. Money, Sydney Hospital photographer, whose assistance is gratefully acknowledged.

References.

BROWN, R. K., and ROBBINS, L. L. (1944), "The Diagnosis and Treatment of Bronchiogenic Cysts of the Mediastinum and Lung", *J. Thoracic Surg.*, 13 : 84.

CONKLIN, W. S. (1950), "Tumours and Cysts of the Mediastinum", *Dis. Chest*, 17 : 715.

HARDY, L. M. (1949), "Bronchogenic Cysts of the Mediastinum", *Pediatrics*, 4 : 108.

HIRSCHFELD, K. (1951), "Tumours and Cysts of the Mediastinum", *Australian & New Zealand J. Surg.*, 21 : 27.

LAIPPLY, T. C. (1945), "Cysts and Cystic Tumours of the Mediastinum", *Arch. Path.*, 39 : 153.

SWEET, R. H. (1950), "Thoracic Surgery", Saunders.

THE INTRABRONCHIAL FOREIGN BODY.

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Inhalation of the Foreign Body.

THE inhalation of a foreign body is usually a dramatic event. There is a choking sensation followed by violent coughing and breathlessness. The ultimate is asphyxia. Indeed, most museums of forensic medicine possess the alarming specimen of a ragged piece of chop wedged firmly in the glottis.

Occasionally there is a history of a slap on the back whilst eating. The slap is unexpected, the victim catches his breath, and down go air and foreign body. In three of our cases this occurred.

The inhalation is an unpleasant experience, but it is one that can be forgotten. Although this is difficult to credit, some patients, when questioned, will emphatically deny ever having inhaled a foreign body, only to recall the incident when confronted with the specimen.

A. (Case 2) was a girl, aged fourteen years, from within whose right bronchus was extracted a mass of vegetable matter resembling a collection of blunt thorns. The Department of Agriculture identified this as the disintegrating remnants of a piece of Norfolk Island pine. A sample of pine was obtained and shown to the patient. At once she remembered both tree and incident. Eight months previously she had been standing under the tree by the edge of the school swimming pool, peeling the end of the piece of pine with her teeth. One of her friends slapped her on the back, and down went the pine. Cotton-wool sandwiches and

reassurance from the teacher did the rest, and everything was forgotten. All was well until the first hæmoptysis three months later.

There are a few upon whom awareness does not dawn even when they are shown the foreign body.

B. (Case 5) had been having repeated hæmoptyses over fourteen months. From within his right bronchus was extracted a knotty, irregular piece of wood half an inch in diameter. He had no recollection of how it came to be there, but at his work (on the cross-cut saws in a sawmill) he was daily bombarded by speedy fragments.

Unawareness can be due to such things as speed of entry, anæsthesia (multiple tooth extractions under general anæsthesia) or drunkenness.

C. (Case 14) had a small piece of dental plate removed from his right bronchus. His plate had been broken in a street brawl three months previously. At the time he had been very intoxicated.

Finally, there are those who give no history of foreign body inhalation, and bronchoscopic examination reveals a normal bronchial tree. However, bronchographic examination reveals a bronchiectatic lobe, and this, when resected, proves to have nurtured a foreign body deep in a basal bronchus.

Symptoms.

Cough.

A persistent cough is the prominent symptom. In the majority of cases it is most distressing. D. (Case 10) stated that "the cough is driving me mad", and her countenance verified the statement. E. (Case 12) stated that "it is so severe I have to hang on for fear of losing consciousness". In many cases it is rasping, racking, distressing and fatiguing in its persistence.

Sputum.

Early there is little sputum; but soon the irritation and inflammation produce a purulent fœtid secretion. There is something characteristic if indescribable about the fœtor—so much so that on two occasions the odour provided the clue to the diagnosis. The amount of sputum varies with the damage to the bronchial wall, and more particularly with the degree of obstructive phenomena in the lobe beyond the blockage.

We have noted that the long, relatively smooth foreign body (for example, a schnapper bone) produces an irritating racking cough with little sputum, whereas the short, thick foreign body (for example, a chop bone) produces a cough with copious sputum. The latter causes more local reaction, more granulation tissue and a greater degree of obstruction, with greater likelihood of obstructive changes beyond.

Hæmoptysis.

In our 20 cases, 11 of the foreign bodies were jagged, irregular pieces of bone. Since such objects cannot help but tear the mucous membrane, hæmoptysis is the rule and in sixteen cases it occurred. Hæmoptysis in the form of staining is the common method of presentation.

Wheeze.

The obstruction to the bronchial lumen by a foreign body gives rise to a wheeze. Sometimes it is the outstanding symptom, as it was in three cases (Cases 8, 12, 17). These three patients had clear-cut histories of foreign body inhalation, and all three produced dirty yellow sputum and had recurrent hæmoptyses. Because of the wheeze a diagnosis of asthma was made and persisted with, despite the patients' insistence as to the presence of a foreign body.

An asthmatic wheeze is generalized. The wheeze of bronchostenosis is localized, or at least maximal over the affected lobe. In the case of foreign bodies, for reasons which we shall elaborate later, the wheeze is to be expected over the lower lobe of the right lung.

It is salutary to remember Jackson's aphorism "all that wheezes is not asthma", especially when the patient remembers the incident of inhalation.

Duration of History.

When a foreign body has been in the bronchus for some time, repeated inflammatory episodes occur in the obstructed lobe. In their clinical aspects they resemble pneumonia and respond as readily to antibiotics.

F. (Case 3), who had a tooth in her right main bronchus for three years, had an average of three attacks of pneumonia each year.

G. (Case 11), a man, aged sixty years, was admitted to hospital in semicoma with a cerebral abscess. Routine examination of his chest revealed signs at the base of his right lung, and an X-ray film of his chest unearthed a three-inch nail in his right bronchus. The cerebral abscess was undoubtedly secondary to the changes in the lower lobe of the right lung. Upon regaining consciousness he described how he had inhaled the nail fourteen years before whilst at his carpenter's bench. In the ensuing fourteen years he had numerous "pneumonic" attacks and many hæmoptyses.

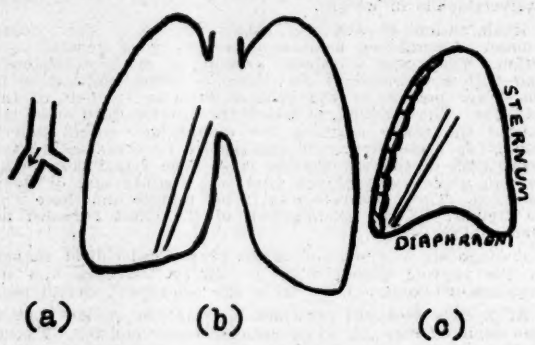

FIGURE I.

We found, provided we excluded Cases 11 and 13, that the average time during which the foreign body remained in the bronchus was eight months. The shortest time was two weeks. This is striking, and illustrates that these cases present rather as problems in chest medicine than as cases of foreign body.

Signs.

Figure I demonstrates two simple anatomical facts. (i) The right bronchus is wider than the left and tends to be the direct continuation of the trachea (Figure I (*a*)). (ii) The posterior basic bronchus, which is the direct continuation of the right bronchus, ends in the region of the cardio-phrenic angle in the postero-anterior X-ray film of the chest, and in the angle between the diaphragm and the vertebræ in the lateral X-ray film of the chest (Figure I (*b*) and (*c*)).

Because of this, gravity encourages lodgement of foreign bodies in the right posterior basic bronchus, so it is over the lower lobe of the right lung that signs will most often be found. What those signs are depends on the nature and degree of the damage to the underlying lung; but it matters little what they are, it is the presence of signs which is of significance. The localized wheeze of bronchostenosis, the crepitations of retained sputum, the atelectasis of complete lobar collapse, even complete absence of all signs, were amongst our more common findings.

X-Ray Examination.

A common question by doctor to radiologist is: "Any evidence of foreign body?" The answer is often: "No evidence of foreign body." In the first place most foreign bodies throw no shadow. In the second, right and left main bronchi are hidden by mediastinal structures. For these reasons it is unlikely that a foreign body will be seen in a plain postero-anterior chest X-ray film.

If we look at any lateral chest X-ray film we note the air-filled trachea and bronchus passing down the centre of the film. It is this lateral X-ray film which is of value. Careful inspection of the lateral X-ray film will sometimes

ILLUSTRATIONS TO THE ARTICLE BY S. LIVINGSTONE SPENCER AND L. R. FINLAY-JONES.

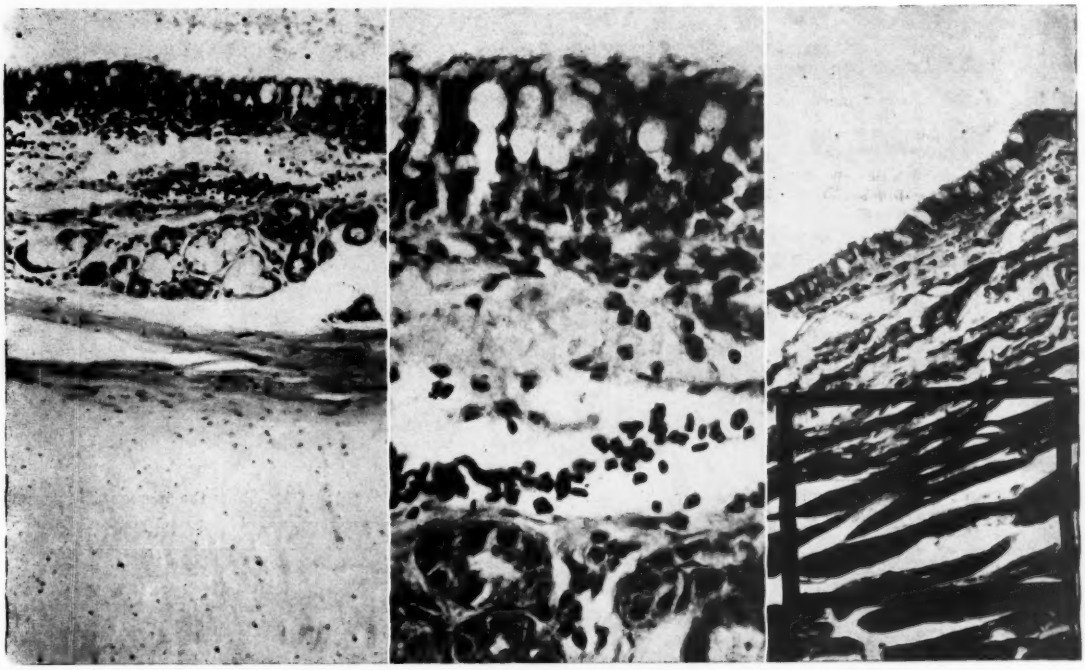

FIGURE I: The cyst wall, with epithelial lining, lymphocytes, glands and hyaline cartilage (× 120). FIGURE II: The epithelium is ciliated, goblet cells are present, and there are mucous and serous glands near the lower border of the field (× 480). FIGURE III: Voluntary muscle fibres are present in the cyst wall in the lower half of the field (× 120).

FIGURE IV.

Showing the cross-striations in the muscle fibres (× 480).

ILLUSTRATIONS TO THE ARTICLE BY A. G. McMANIS AND HARRY M. WINDSOR.

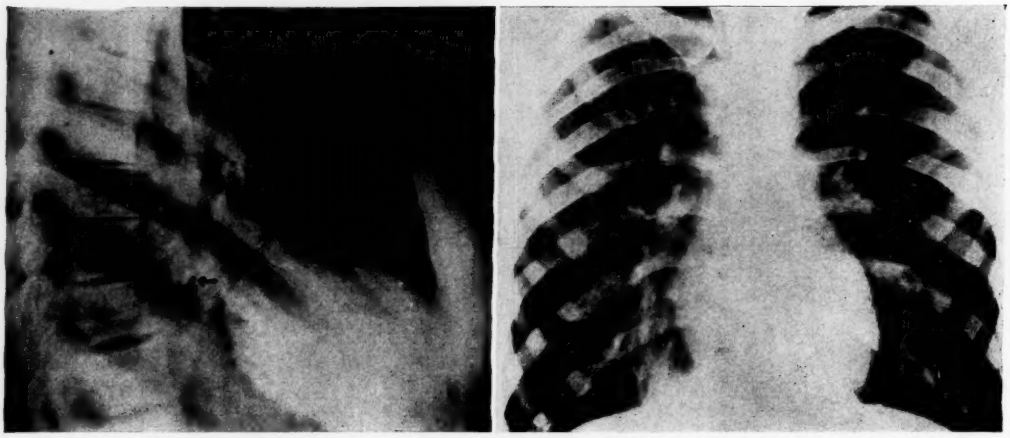

FIGURE II. FIGURE III: Postero-anterior skiagram of chest (Case 9—barley grass) to show the increased lung markings in the right cardio-phrenic angle.

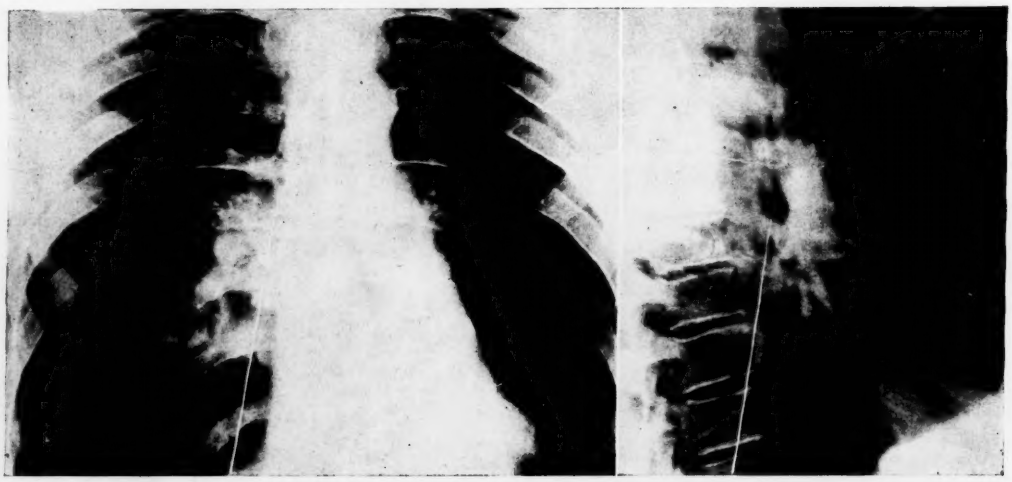

FIGURE IV: Postero-anterior skiagram of the chest with a marker placed in the line of the posterior basic bronchus, to show the relative position of this bronchus. FIGURE V: Lateral skiagram of the chest with a marker placed in the line of the posterior basic bronchus, to show the relative position of this bronchus.

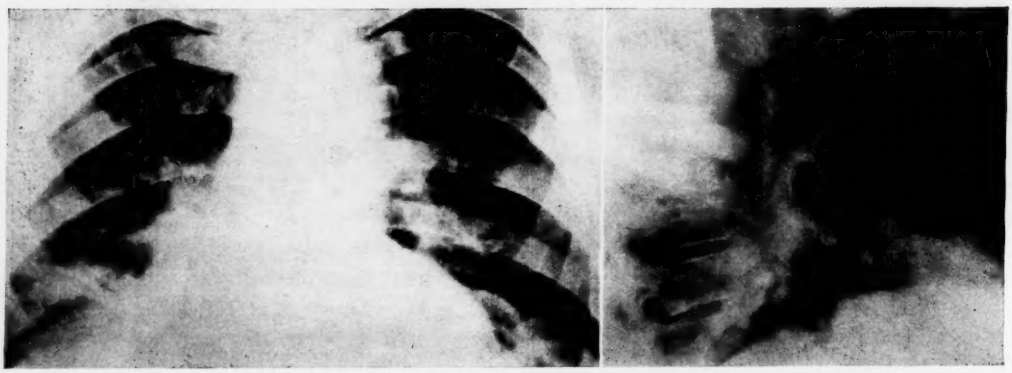

FIGURE VI: Postero-anterior skiagram of the chest (Case 20—bone), to show the typical sites of the opacities in the line of the posterior basic bronchus. FIGURE VII: Lateral chest skiagram (Case 20—bone), to show the typical sites of the opacities in the line of the posterior basic bronchus.

ILLUSTRATIONS TO THE ARTICLE BY HOWARD WILLIAMS.

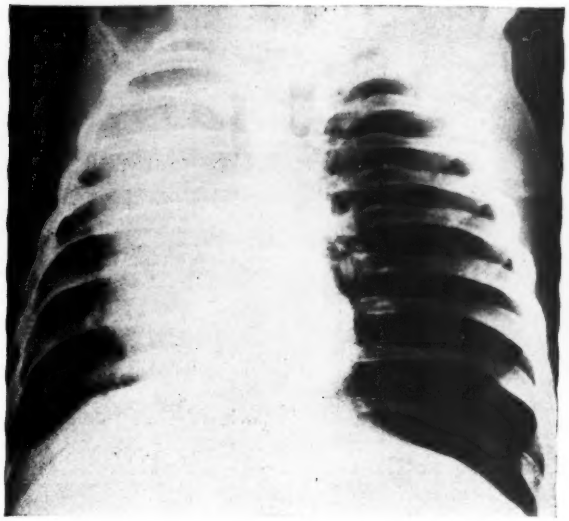

FIGURE I.

Radiograph of the chest showing mediastinal displacement to the right, depression of the left dome of the diaphragm and emphysema of the left lung.

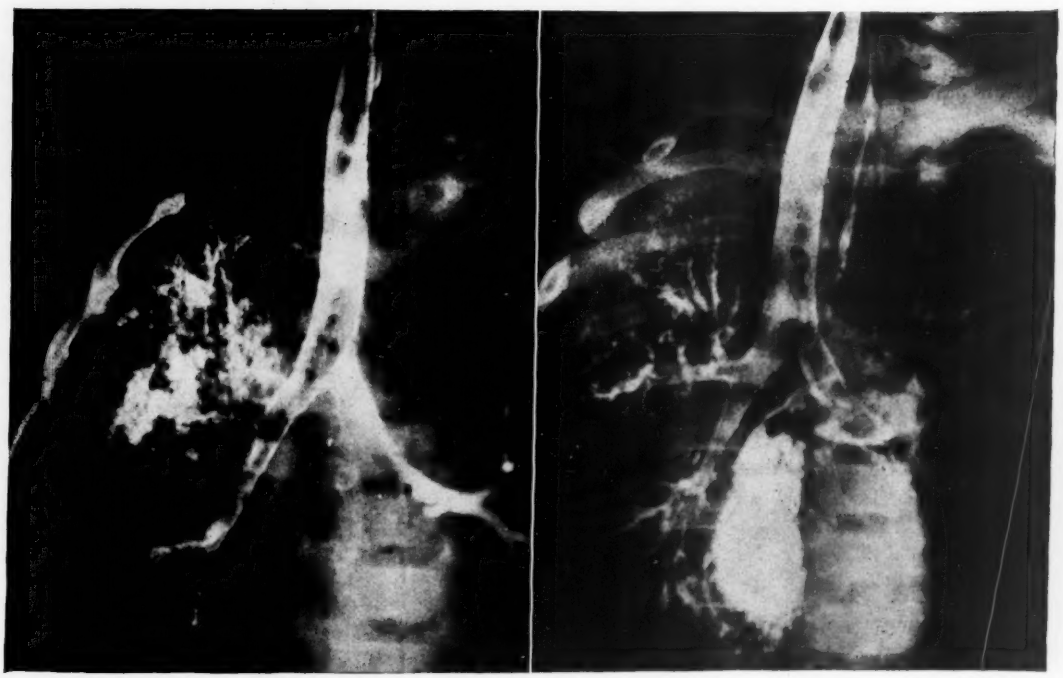

FIGURE II.

Bronchogram showing narrowing of the left bronchus from compression by the bronchogenic cyst.

FIGURE III.

Bronchogram showing bronchogenic cyst filled with lipiodol and communicating with the left bronchus by a stalk. Note that following bronchoscopy the left bronchial lumen is now of normal calibre.

ILLUSTRATIONS TO THE ARTICLE BY K. REYE, M.B., B.S.

FIGURE I. (× 100.)

FIGURE II. (× 400.)

ILLUSTRATIONS TO THE ARTICLE BY R. V. SOUTHCOTT AND MALCOLM FOWLER.

FIGURE I: A celloidin section from the upper half of the left cerebral hemisphere (cut sagittally), showing the cavity and the perivascular and diffuse demyelination. Weil's myelin sheath stain (× 1·5). FIGURE II: Perivascular demyelination in the white matter of a gyrus. Weil's myelin sheath stain (× 18). FIGURE III: Scavenger cells in an area of perivascular demyelination. Hæmatoxylin and eosin stain.

TABLE I.
Summary of Cases.

Case Number.	Incident of Inhalation.	Symptoms.	Signs.	X-Ray.	Foreign Body; Period *in Situ*.	Method of Removal.
1	Remembered incident.	Cough, sputum, wheeze, hæmoptysis.	Collapse of lower lobe of right lung. Localized wheeze.	Collapse of lower lobe of right lung.	Chop bone, 4 months.	Bronchoscopy.
2	Did not remember incident.	Recurrent hæmoptysis.	Crepitations at base of right lung.	Increased markings at right cardio-phrenic angle.	Piece of Norfolk Island pine, 6 months.	Bronchoscopy.
3	Did not remember incident. Teeth extraction under general anæsthesia.	Cough, sputum, recurrent pneumonia.	Collapse of lower lobe of right lung.	Collapse of lower lobe of right lung. Foreign body seen in lateral X-ray film.	Tooth, 3 years.	Bronchoscopy; later lobectomy.
4	Did not remember incident. Teeth extraction under general anæsthesia.	Cough, sputum, hæmoptysis.	Crepitations at base of right lung.	No abnormality detected.	Tooth, 6 months.	Bronchoscopy; later lobectomy.
5	Did not remember incident.	Cough, sputum, hæmoptysis.	Collapse of lower lobe of right lung.	Collapse of lower lobe of right lung.	Piece of wood, about 14 months.	Bronchoscopy; later lobectomy.
6	Remembered incident.	Cough, sputum, hæmoptysis.	Crepitations at base of left lung.	Increased markings at base of left lung.	Chop bone, 2 years.	Coughed it out.
7	Remembered incident.	Cough, sputum, hæmoptysis.	No signs.	No abnormality detected.	Chop bone, 2 months.	Coughed it out.
8	Remembered incident. Could not convince doctor.	Cough, wheeze.	Localized right basal wheeze.	Increased marking at base of right lung. Foreign body seen in lateral X-ray film.	Chop bone, 1 month.	Bronchoscopy.
9	Remembered incident.	Cough, sputum, hæmoptysis.	Crepitations at base of right lung.	Increased markings at right cardio-phrenic angle.	Barley grass, 12 months.	Coughed it out. Urgent lobectomy for severe hæmotyses.
10	Remembered incident.	Cough, sputum, wheeze, hæmoptysis.	Localized wheeze at base of right lung.	Increased markings in right cardio-phrenic angle. Foreign body seen in lateral X-ray film.	Schnapper bone, 2 weeks.	Bronchoscopy.
11	Remembered incident.	Semi-coma; recurrent hæmoptysis.	Collapse of lower lobe of right lung.	Collapse of lower lobe of right lung. Foreign body seen in lateral X-ray film.	Nail, 14 years.	Nil.
12	Remembered incident; could not convince doctor.	Cough, sputum, hæmoptysis, wheeze.	Crepitations and wheeze, base of right lung.	Increased markings, right cardio-phrenic angle.	Chop bone, 8 months.	Bronchoscopy.
13	Did not remember incident.	Recurrent hæmoptysis.	No abnormality detected.	Opacity in left lower zone.	Chop bone?, many years.	Coughed it out.
14	Did not remember incident; drunken brawl.	Recurrent hæmoptysis.	No abnormality detected.	No abnormality detected.	Broken dental plate, 3 months.	Bronchoscopy.
15	Remembered incident.	Cough, sputum, hæmoptysis.	Crepitations, base of right lung.	Increased markings, right cardio-phrenic angle.	Chop bone, 4 months.	Bronchoscopy.
16	No history of inhalation obtained.	"Wet" cough.	Generalized crepitations.	Localized emphysema, middle lobe.	Peanut.	Bronchoscopy.
17	Remembered incident; could not convince doctor.	Cough, sputum, hæmoptysis, wheeze.	Crepitations and wheeze, base of right lung.	Increased markings, right cardio-phrenic angle.	Chop bone, 2 weeks.	Bronchoscopy.
18	Did not remember incident.	Cough, sputum.	No abnormality detected.	No abnormality detected.	Button.	Bronchoscopy.
19	Remembered incident.	Cough, sputum, hæmoptysis.	Crepitations, base of right lung.	Increased markings, right cardio-phrenic angle. Foreign body seen in lateral X-ray film.	Chop bone, 7 months.	Bronchotomy.
20	Remembered incident.	Cough, sputum, hæmoptysis.	Crepitations, base of right lung.	Increased markings, right cardio-phrenic angle.	Chop bone, 2 months.	Bronchoscopy.

reveal the tell-tale opacity in the line of the bronchus. We noted it in five of our cases (three bones, one tooth, one nail). It was particularly striking in Case 3, that of F. The tooth with its broken attenuated roots was clearly visible lying in the bronchus (Figure II).

What is found, however, is evidence of the obstructive changes. This evidence ranges from a few increased markings (Figure III) to total lobar collapse.

If we again consider Figure I, and Figures IV and V, we shall note that the posterior basic bronchus—the bronchus into which gravity takes the foreign body—lies in the cardio-phrenic angle in the postero-anterior X-ray film, and in the costo-diaphragmatic angle in the lateral X-ray film. These are the sites in which the opacities are to be expected, and in which they were present in 14 of our cases (Figures VI and VII).

Absence of opacity does not exclude foreign bodies. C. (Case 14) had a perfectly normal chest X-ray picture. Nevertheless, he had a non-obstructing piece of dental plate in his right bronchus.

The infrequency of a shadow on the left side is in keeping with the small percentage of foreign bodies found on that side—two cases out of 20 (Cases 6 and 13) in our series.

Baby H. (Case 16), a child aged two and a half years, demonstrated the interesting but uncommon finding of localized obstructive emphysema. The cause was a peanut in the right bronchus.

Management.

The management is bronchoscopic examination and removal of the foreign body. Apart from meat and fish bones, we have encountered wood, teeth, broken dental plate, Norfolk Island pine, barley grass, a nail, a button and a peanut.

It is not the purpose of this paper to describe the many little tricks associated with the endoscopic removal of these objects, many of which have a diameter half as large again as the bronchoscope. Suffice to say that once they are out recovery is rapid and in most cases complete.

Chop bones are often broken up by the battering they receive on the butcher's block, so inhalation of a chop bone may mean inhalation of a number of fragments held together by muscle. This has been of practical significance.

Mrs. J. (Case 1), a woman, aged sixty-three years, knowingly inhaled a chop bone. It was removed by us at bronchoscopy two months after its entry. Her symptoms persisted until a month later, when she coughed out the remaining piece.

It is very easy to overlook a residual piece of bone owing to the bleeding and trauma consequent upon removal of the initial piece. Likewise it is easy to overlook the foreign body completely at bronchoscopy if it is embedded in friable granulation tissue, as occurred in Case 12. This patient had an unsuccessful bronchoscopy one month before the piece of bone was ultimately removed.

We were able to remove the foreign body through the bronchoscope in 14 of our cases. Four coughed it out. In the case of G. (Case 11) no attempt was made to remove the nail.

Bronchotomy.

K. (Case 19) had a large irregular piece of bone firmly anchored in and completely obstructing his right bronchus. We could not dislodge it. Each attempt caused considerable bleeding and trauma. A thoracotomy was carried out, the bronchus opened and the foreign body removed. A few sutures in the bronchus completed a relatively simple procedure.

Definitive Management.

If a foreign body has been *in situ* for any length of time, it is a corollary that irreversible obstructive changes will have occurred. If this is the case the symptoms, although relieved, will not disappear after its removal. In all cases in which symptoms persisted we carried out further bronchoscopy and bronchography. In three a hopelessly destroyed residual lobe was found, and in each a lobectomy was carried out (Cases 3, 4 and 5).

On one occasion (Case 9—Windsor, 1951) we were confronted with the problem of massive recurrent hæmoptyses. An urgent lobectomy was necessary for the control of the bleeding.

Medico-Legal Considerations.

We are all familiar with the case of patient *versus* dentist. Most of us remember being party to a quick ethyl chloride or gas anæsthetic, whilst the dentist extracted a full upper or lower set or even both. A pharynx full of blood, a dish full of teeth and a blue struggling patient were the characteristics. It is little wonder that teeth frequently dropped through the glottis.

There is hardly excuse for such carelessness any longer. With excellent anæsthesia readily available such accidents are completely preventible. F. (Case 3) lost the lower and middle lobes of her right lung as the result of such an accident. Surely, since such negligence is indefensible, right is on the patient's side.

A further medico-legal aspect is illustrated by Case 5. The foreign body in this case was a ragged, irregular piece of wood, and the patient had no recollection of the inhalation. At his work in the sawmill he was often struck by flying fragments of wood. He sued for and won his workers' compensation. It is thought that the affirmative answer to Mr. Justice Dignam's question, "Is it possible for a man to inhale such a thing and not know it?", may have influenced the decision.

Conclusion.

We should like to stress the following points:

1. Failure to obtain a history of inhalation does not exclude the presence of a foreign body.

2. A history of recent onset of cough with hæmoptysis and a shadow in the right cardio-phrenic angle ought to suggest foreign body.

3. A bronchostenotic wheeze can easily be confused with an asthmatic wheeze.

4. A foreign body will rarely be obvious as such in a plain X-ray film of the chest.

Reference.

WINDSOR, H. M. (1951), "Massive Hæmoptysis from Intrabronchial Foreign Body", M. J. AUSTRALIA, 1: 694.

Reports of Cases.

BRONCHOGENIC CYST IN INFANCY, CAUSING RESPIRATORY OBSTRUCTION.[1]

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BRONCHOGENIC CYSTS of the mediastinum are uncommon and very few cases have been recorded in infants or children. Hardy (1949), in a review of the literature, stated that 82 cases had been reported and only three were in infants or children. He reported the case of a baby, aged eight months, in whom the condition was successfully diagnosed and surgically treated; Maier (1950) and Adams (1951) have also recorded similar successes, the former in a child aged two and a half years, the latter in an infant. Carlson (1942), Marshall and Cookson (1943), and Troendle, Sanford, Lo Presti, Anderson and Rice (1950) record three separate cases in which a bronchogenic cyst caused death from obstruction to the trachea and main bronchi, but the cause was unsuspected during life. I have seen one post-mortem examination which revealed an unsuspected bronchial cyst in a baby aged ten months, who had cyanotic attacks and wheezing from birth. This cyst was 2·5 by 3·0 centimetres in area, contained clear mucoid fluid, was wedged in the bifurcation of the trachea between the bronchi, and had caused compression of the trachea and both bronchi. It is only by a better appreciation of the clinical features that may arise from a bronchial cyst, and by complete investigation in suspected cases, that the tragedy of the post-mortem diagnosis of this surgically remediable lesion will be avoided. It is for this reason that this case is reported.

Clinical Record.

A male child was admitted to the Royal Children's Hospital at the age of five months on September 22, 1952, with a history of intermittent dry cough and persistent wheezy breathing since the first few days of life. He was an illegitimate child and no details are known of the pregnancy. Labour was normal and the birth weight was five pounds twelve and a half ounces. When he was ten days old he was transferred to a babies' home, where the nurses noted that his breathing was wheezy during both phases of respiration. At times a short dry cough was heard, but no other symptoms were noted. He took his

[1] This patient was shown at a meeting of the Melbourne Pædiatric Society on August 12, 1953.

feedings well, gained weight steadily and weighed twelve and a half pounds at five months.

Examination revealed him to be a well-nourished, alert baby with a good colour. The breathing was wheezy in character, the expiratory phase being longer than the inspiratory phase. The thoracic cage bulged anteriorly, more on the left side than on the right, and the costal margins of both sides of the chest and the suprasternal and supraclavicular spaces retracted during inspiration. The heart was displaced to the right, the percussion note over the left lung was hyperresonant and the breath sounds were poor, while those over the right lung were normal. However, the percussion note over the right lung anteriorly was impaired. No other abnormality was detected on clinical examination. Radiological examination demonstrated that the heart and mediastinum were displaced considerably to the right, the left lung was emphysematous, and the left side of the diaphragm was low in position (see Figure I). It was thought that the upper lobe of the right lung was collapsed. The Mantoux test failed to produce a reaction with 1 in 1000 old tuberculin. The blood examination revealed no abnormality. Bronchoscopic examination revealed that the left main bronchus was narrowed to a crescent-shaped slit one centimetre from the bifurcation of the trachea. This narrowing seemed to be due to bulging of the posterior and medial portion of the bronchial wall into the lumen. The bronchoscope could be passed easily and without resistance through this narrowed slit, and the orifices of the upper lobe and of the dorsal and lower lobes were identified as normal. The right bronchus and divisions to upper, dorsal and middle lobes were normal. A bronchogram (see Figure II) confirmed the narrowing of the left bronchus and demonstrated that the narrowed portion extended for 0·75 centimetre, and then the bronchus widened to a normal calibre. The left side of the bronchial tree could not be adequately filled with radio-opaque oil owing to emphysema of the left lung, while the right side of the tree was normal. The cause of the bronchial narrowing was considered to be maldevelopment of the cartilage of this portion of the left bronchus.

During the next three months the infant's symptoms and clinical and radiological signs persisted unchanged, and he continued to feed well and gain in weight (weighing fourteen pounds at nine months). Another bronchoscopic examination at the age of nine months revealed the same left bronchial obstruction. However, after the bronchoscope had been passed through the narrowed left bronchus a considerable amount of clear, mucus-like fluid was aspirated, but it was not possible to determine the source of this fluid. A bronchogram disclosed a large oval cyst measuring 2·0 by 3·5 centimetres filled with air and lipiodol, situated in the posterior mediastinum behind the heart and in front of the œsophagus, and communicating with and compressing the left bronchus (see Figure III). It was now very evident that a bronchogenic cyst was the cause of the infant's symptoms and signs. After discharge of the contents of the cyst after bronchoscopic examination the wheezing became less and the air entry into the left lung better, and radiologically the heart moved partially toward the normal position. Improvement was not maintained, for after two weeks the child's symptoms and signs recurred.

On February 11, 1953, when the child was aged ten months, a bronchogenic cyst approximately 2·0 by 4·0 centimetres in diameter was removed by Mr. Russell Howard. The cyst was found to be adherent to the œsophagus and was intimately connected with the posterior aspect of the left bronchus one centimetre from the trachea. The musculature of the bronchus and cyst was a continuous sheet of tissue. Dissection of the cyst left a deficiency in the cartilage and muscular structure of the bronchial wall, which was repaired by muscle and fibrous tissue of that part of the cyst wall adjacent to the bronchus. The cyst contained mucoid fluid and air.

Dr. Alan Williams reported that the cyst measured 3·5 by 2·5 by 2·5 centimetres, was covered with loose connective tissue, and had a firm wall 3·0 millimetres thick which felt as if it contained cartilage. The inner mucosal surface was wrinkled. Microscopic examination of the wall revealed an inner mucosal lining consisting chiefly of stratified squamous epithelium with several small areas of pseudostratified columnar epithelium. The submucosal layer was comprised of a loose areolar tissue in which were scattered mucus-secreting glands and groups of inflammatory cells. The main thickness of the wall was comprised of two layers of involuntary muscle running across each other, together with fibrous tissue. Several pieces of cartilage were noted in this layer. The specimen was considered to be a typical bronchial cyst.

Post-operative convalescence was complicated by bronchitis and bronchiolitis, but recovery was rapid. Since then the child has continued to thrive and develop normally and has been entirely free of symptoms. Examination at the age of sixteen months revealed him to be a healthy normal baby, with no abnormal physical signs. Radiological examination of the chest indicated that the heart had not completely returned to the normal position, but the left lung was no longer emphysematous and the left side of the diaphragm was in the normal position. The final result is more than satisfactory, as this baby has been adopted and now has a good home.

Discussion.

Bronchogenic cysts probably originate from a defect in budding of the primitive lung, but the cause is unknown. These cysts are usually found in the posterior mediastinum behind the trachea or bronchi and in front of the œsophagus, but may occasionally be situated in any position in the mediastinum. They tend to grow slowly and frequently do not cause symptoms until adult life and often only in later years. They may never cause symptoms and be discovered only during a routine post-mortem examination.

The pædiatrician should appreciate that if symptoms occur early they generally date from birth or shortly after, and are almost without exception due to compression of the trachea or bronchi with respiratory obstruction. Vomiting due to œsophageal compression rarely occurs, probably owing to the mobility of the œsophagus and to the ease with which fluids and soft semi-solids slip down a narrowed lumen. The predominant symptom from respiratory obstruction is wheezy breathing. If obstruction is severe, cyanotic attacks may occur, and if infection develops behind the obstruction the signs of bronchitis and lung infection may mask the obstructive lesion. A spasmodic cough is common, owing to bronchial compression.

Diagnosis is difficult. Plain skiagrams will help to identify the cyst only if it projects from the mediastinum into the lung field or if it casts a shadow sufficiently dense to differentiate it from other structures in the mediastinum. Indirect evidence may be obtained from tracheal, bronchial or œsophageal displacement and narrowing. The diagnosis will be made more frequently if every baby with chronic cough and wheeze is radiologically examined and those in whom a bronchial or tracheal obstruction is suspected are subjected to bronchoscopic and bronchographic investigation. Bronchial asthma should be accepted as the cause of an infant's cough and wheeze only when other causes of bronchitis, bronchiolitis, and narrowing of the trachea or major bronchi have been excluded.

Summary.

A case of bronchogenic cyst in an infant, aged ten months, has been successfully treated. The clinical features were persistent wheezing and spasmodic cough since birth, with emphysema of the left lung and gross mediastinal displacement due to left bronchial obstruction.

Acknowledgement.

I wish to thank Dr. Stanley Williams for his courtesy in allowing me to report this case.

References.

ADAMS, F. H. (1951), "Unusual Case of Bronchiogenic Lung Cyst Simulating Dextrocardia", *J. Pediat.*, 39: 483.

CARLSON, H. A. (1943), "Congenital Cysts of the Mediastinum. Report of 3 Cases including Gastric Cyst", *J. Thoracic Surg.*, 12: 376.

HARDY, L. M. (1949), "Bronchogenic Cysts of the Mediastinum", *Pediatrics*, 4: 108.

MARSHALL, P. C., and COOKSON, H. A. (1943), "Report of a Case of Tracheo-Bronchial Cyst", *Lancet*, 1: 305.

MAIER, H. C. (1950), "Tracheal Compression from Bronchogenic Cyst in Esophageal Wall", *Am. J. Dis. Child.*, 80: 423.

TROENDLE, F. J., SANFORD, M., LO PRESTI, J. M., ANDERSON, R., and RICE, E. C. (1950), "Clinico-pathologic Conference: Bronchiogenic Cyst", *Clin. Proc. Child. Hosp., Wash.*, 6: 165.

BASAL CELL CARCINOMA OCCURRING IN CHILDHOOD.

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THE occurrence of newgrowths of the skin in childhood is rare. The following is the report of a basal cell carcinoma excised from a child aged five years.

Clinical Record.

A girl, aged five years, presented with a lump in the right naso-labial furrow. Her mother said it had been growing there since it first appeared six to twelve months previously. There was no history of injury or burn. The family lived on the edge of an almost treeless blacksoil plain in north-west Queensland; even the grass on this plain has lost its green for the longest part of the year and therefore is particularly glaring. Next to the child's home was a fuel oil depôt where petrol, oils, kerosene *et cetera* were stored or poured into smaller tins for sale. The patient was a constant companion of the owner of the depôt and went with him when he delivered petrol *et cetera*.

The child's grandmother, living close by, had had treatment for a number of rodent ulcers about the face. The child's father stated he had had a "pimple" in his left naso-labial furrow, but closer to the nose than in the child's case; it had been present for about twenty years (first noticed by him when he was about twenty-five years of age) without trouble until a barber nicked it with a razor, after which it would not heal. Records consulted showed that the father was treated with implant of radon seeds for a "rather large rodent ulcer", and that four other areas were treated by radon plates. The child had four older brothers who were not affected; the mother was also clear of any sign of rodent ulcers. The family extraction was Irish and English.

The child's complexion was fair and somewhat freckled. The lump was about midway along the naso-labial fold, attached to the skin, smooth and rounded. The epithelium was intact, but appeared thinned by stretching over the lump; on macroscopic examination of a section the tumour was solid. The initial pathological report stated that the tumour was a typical basal cell carcinoma, that Ewing referred to one in a child, aged fourteen years, and that its occurrence on the naso-labial fold suggested the possibility of origin at the site of malformation.

The wound healed well; the child was observed for about six months and there was no recurrence, and no new suspicious areas were detected.

Differential Diagnosis.

On microscopic examination there would appear to be no doubt that this is a basal cell carcinoma. Basal cell carcinomata occurring in children have been described previously. They are known to occur in *xerodermia pigmentosum* and in *epithelioma adenoides cysticum* (Brooke). However, this patient shows none of the stigmata of *xerodermia pigmentosum*, and in a number of ways the condition is unlike *epithelioma adenoides cysticum*. Sweat gland tumours need special mention.

Xerodermia Pigmentosum.

Sequeira defines *xerodermia pigmentosum* as a "rare affection characterised by permanent freckling, telangiectasis, atrophy of the skin, and the formation of warty tumours which frequently become malignant—the disease may affect several members of a family—it does not appear to be hereditary, the fact that the patients rarely survive puberty probably explaining this". Molesworth noted that "perhaps the commonest accompaniment of rodent disease is a tendency on the part of the skin to freckle and in *xerodermia pigmentosum* such a patient exhibits what is only the extreme degree of susceptibility of which the red-haired, freckled individual represents the next grade".

A "freckle count" of the head and neck was begun on the children of the patient's age at her school. However, without counting far it was obvious that she was no more freckled than the average, and that a number of children had very many more freckles. No children were without freckles.

Epithelioma Adenoides Cysticum (Brooke).

Epithelioma adenoides cysticum (Brooke) is a congenital and familial affection of the hair follicles or perhaps the sweat glands. The tumours appear in childhood. The seats of election are the central third of the face, the root of the nose, the nostrils and adjacent parts of the cheeks, upper lip and chin. It is commoner in females than in males. Goldsmith and Freudenthal reported the case of a boy, aged seven and a half years, with onset at five years. The lesions were on the face especially and around the eyes and also on the neck.

Paul and Inglis (1927) reported the case of a female, aged thirty-one years; the condition first manifested itself on the face when the patient was about two years old. Since that time the lesions had gradually increased as regards the number of tumours and their size; at the age of thirty-one years there were numerous small tumours scattered over the face.

Both these groups of authors describe multiple lesions. While it is quite possible for the child in the present case to develop further lesions later on, this was a single lesion, and no new ones had arisen up to six months after the time of excision.

Sequeira writes: "Dr. Savatard believes that single Brooke tumours are often mistaken for common moles."

Warvi and Gates divide cystic epithelial tumours into two groups, as follows:

> (1) Those which are multiple, with certain clinical and histologic characteristics suggesting a congenital origin.
>
> (2) Those which are primarily tumours of middle age and may be classed with the nonkeratinizing tumours.
>
> There is no sharp dividing line between the two groups either on a clinical or a histologic basis. Nonkeratinizing Epithelial Tumours of the skin of which the basal cell Carcinoma is a frequent form, are prone to cystic degeneration of both Tumour and the Stroma. Such tumours have often been confused with the true cystic tumours of sweat glands.

Solid Carcinoma of Sweat Glands.

Carcinoma of sweat glands in their solid form "may resemble basal cell carcinomata, but the pattern of the growth is quite different and the alveoli lined by cuboidal epithelium embedded in solid cell masses makes the origin clear" (Gates, Warren and Warvi, 1943). However, they mention that in undifferentiated growths it may not be possible to distinguish the tumour from basal cell carcinoma. These sweat gland tumours are usually noted in middle life; but some patients give a history of their presence since early childhood; this may be the result of malignant change in a previously benign growth.

Molesworth has noted finer cavities and lace pattern with epithelial connexion.

Histological Features.

The outstanding feature of *epithelioma adenoides cysticum* is "cysts many of which contained horny lamellae" (Goldsmith and Freudenthal's case). Inglis pointed out the following findings in his sections: (i) accumulation of keratin showing concentric lamination; (ii) hyaline material; (iii) cysts; (iv) retrograde changes with phagocytic giant cells. He held (i) and (ii) to represent an attempt at formation of hair and sebaceous glands.

In the section presented (Figure I), features (i), (iii) and (iv) were absent. There is an area shown in the high-power photomicrograph (Figure II) which may be early formation of hyaline material. The section does show pseudo-glandular formation (lower margin of the low-power photomicrograph), and in another area (not shown) there is an appearance of the laciform pattern found in basal cell carcinomata. The pattern of the growth does not suggest to me a sweat gland tumour.

Thus, as far as one can say, this tumour resembles more the basal cell-growths of the superficial type which are so common in Queensland. It does not signify that the tumour did not arise from subepidermal structures, for as Willis states: "No sharp separation between basal-cell growths of superficial and deep types is possible; growths similar in structure and behaviour may arise from basal cells in any part of the epidermis and its appendages."

Discussion.

If, as it appears to me, this is more like the types of tumour found after exposure to sunlight over a long period, then it is of interest to see if we can discover any factors which may have led to its early development, in contrast to the usually long latent period usually found with rodent ulcers. Factors to be considered are age, site, heredity, sunlight, dryness, and other carcinogens.

Age of Onset.

Review of the Literature.

H. W. Dargeon's series of patients had cancer of the skin of the face in three instances and cancer of the upper lip in two: ". . . the youngest at the age of twenty months and the oldest in a boy of eleven. The histologic form . . . includes squamous carcinoma, basal cell carcinoma, and spindle-cell carcinoma. . . . Two cases of the series were *xeroderma pigmentosum*".

Molesworth, in his comprehensive review of rodent ulcer, states that the youngest patient to come under his own notice was a youth, aged twenty years, whose skin showed every mark of exposure. Further, in discussing a microscopic section from a tumour of a three year old child, he states that "the extreme youth of the patient seems to exclude rodent formation as an interpretation of the main part of the growth".

French (1948), in his "Index of Differential Diagnosis", states: "Rodent ulcer differs from *lupus vulgaris* in not being a disease that starts in childhood."

In Wakeley and Childs's series (1949), the youngest patient was aged forty-seven years.

Willis quotes Shrek and Gates as stating that the commonest age of onset is sixty-six years.

Personal Inquiries.

It was thought that as these growths are common in Australia, and as "sunny Queensland" is in the more tropical part of this continent, basal cell carcinoma might be more common here in childhood than world literature suggests. However, the following information was obtained.

The "Annual Report of the Queensland Radium Institute" (1952) showed that from January, 1940, to December, 1951, 10,081 persons were treated for the basal cell type of carcinoma. Of these, 15 were aged under twenty years, and only 1160 or 11·5% were under forty years old. The exact ages of these patients were not available.

A Brisbane pathologist of long standing reported that of many thousands of cases of skin cancer he had seen only one in a child—squamous epithelioma on the dorsum of the foot of a very young child (aged ten years or under).

The superintendent of the Brisbane Children's Hospital reported that in his nine years there he had not seen one such tumour in a child, nor had a pædiatrician visiting there for a quarter of a century.

A number of other country doctors and doctors treating these conditions were questioned, but no similar case of basal cell carcinoma in a child was disclosed.

Site.

It was wondered whether a higher proportion of these lesions occured on the face in Queensland.

The report of the Queensland Radium Institute (January, 1940, to December, 1951) showed that 736 out of 11,768 basal cell carcinomata occurred in other places than on the head and neck—that is, 93·8% occurred on the head and neck.

Wakeley and Childs (1949) described 27 cases (of a series of 237) which occurred elsewhere than on the face and scalp. Four of these were on the neck; that is, 90·3% occurred on the head and neck in an English series.

Significance of the Naso-Labial Furrow.

It should be made clear that the naso-labial furrow, crease or fold here mentioned refers to that line joining the angle of the mouth to the ala of the nose. It does not refer to the elevation bounding the philtrum, which elevation corresponds with the *musculus nasolabialis*, the medial band of the *musculus orbicularis oris* connecting the upper lip to the back of the septum of the nose. This elevation is much nearer to a site of embryological fusion (the mid-line) than the naso-labial furrow.

It has been suggested in the initial pathological report that the embryonic fusion lines may have some significance in the formation of this and similar tumours. Boyd mentions misplaced cell groups from the epidermis as one possible site of origin of rodent ulcers. "The fact that they are often situated along the line of embryonal fissures on the face supports an origin from misplaced cell groups." However, many would not agree with these statements, and it seemed worthwhile to consider the modern concept of the cell rest theory, the embryological lines of fusion of the face, and the anatomy and special features of the naso-labial furrow.

Cell Rest Theory.

There has been a lessening in the popularity of the cell rest by hypothesis now that it has become "clear that all cells are 'cell rests'—that is, all cells have retained in some measure their primary capacity for proliferation and differentiation" (King, 1948). In "Studies in Pathology" (1950), Willis states that great transformations of cellular structure are possible in most tissues, and he follows this statement with numerous examples of metaplasia.

Embryological Lines of Fusion.

The question of the exact sites of embryological fusion does not appear to be settled. The older teaching was that the lateral hare lip represented imperfect fusion of the medial nasal portion of the fronto-nasal process and the maxillary process on that side (McGregor, 1946).

Frazer (1946) makes the following statement:

> The fronto-nasal process . . . very soon gets covered by maxillary mesoderm reaching it from behind along the roof of the buccal cavity, and from it in front after the fusion between it and the main maxillary mass. This last invasion covers it completely from the front; forming the upper lip and probably the alveolus of the premaxilla, and actually the only part of the original fronto-nasal process to reach the surface is the small incisive tubercle behind the upper central incisors.

A rare lateral or oblique congenital facial cleft, which extends from the angle of the mouth up the cheek to the eye, seems to be the nearest line of possible "non-fusion"

to the naso-labial fold. Even prior to Frazer's observations, this cleft was not explainable on the theory of the lines of fusion then held, fusion of the lateral nasal process and the maxillary process usually being held to be along the edge of the nose; though Arey maintains that this oblique cleft is indeed due to a failure of union between a maxillary process and the fronto-nasal process.

Anatomy.

With regard to skin creases, Clark (1946) makes the following statement:

> These are flexure lines—or "skin joints"—about which the skin can be easily folded in association with movements of the parts which it covers. A section through a flexure line shows that here the skin is usually adherent to the underlying deep fascia. In the region of face and neck, with the movement of facial expression, flexure lines appear momentarily in the skin at right angles to the pull of the fibres of cutaneous muscles. In other words, their pattern is determined by the arrangement of the underlying musculature. With advancing age, as the result of the progressive loss of elasticity of the skin, these creases become established as permanent wrinkles.

"Gray's Anatomy" describes the *musculus levator labii superioris* assisting the *zygomaticus minor* to form the naso-labial furrow.

Cunningham mentions that both muscles are inserted into the skin of the naso-labial groove. The direction of the fibres of these two muscles is approximately at right angles to the naso-labial groove.

Sebaceous Glands.

Sequeira noted that large sebaceous glands are seen on the nose and in the naso-labial furrows. Molesworth stated that rodent tumours show a special predilection for the middle third of the face, which is an area occupied by the most active sebaceous glands.

Willis writes:

> The excretion of carcinogenic substances by the sebaceous or sweat glands must be considered as a possible factor in the causation of skin tumours. This possibility . . . appears particularly plausible in cases of multiple subepidermal basal cell growths of predominantly glandular origin, as in Brooke's disease or turban tumours. The association of degenerated parasites (Demodex) obstructing the glandular orfices, with some basal-cell skin tumours, may not be purely fortuitous; it may be that the obstruction causes basal accumulation of carcinogens which are constantly being discharged in small amounts by the skin glands.

Molesworth mentions a tumour which may be a solitary benign cystic epithelioma generally in the neighbourhood of the naso-labial fold.

Also Sequeira gives the following description: "Adenoma Sebaceum is a congenital overgrowth of the sebaceous glands—affecting the middle third of the face, and especially the naso-labial furrows."

It would seem from these that the sebaceous glands are of some special significance in the middle third of the face.

Mechanical Considerations.

Mechanically one would expect such skin creases to catch and retain more particles of dirt and carcinogens. Ross noted that both pitch warts and epitheliomata were commonest on the face, particularly in the neighbourhood of the nostrils. He did not make any special reference to skin creases.

Amount of Subcutaneous Tissue.

Trethowan (1952) stated that "in many commonly affected areas there was relatively little subcutaneous tissue". As was quoted from Clark above, the skin is usually adherent to the underlying deep fascia at a flexure line.

Conclusion.

Thus it would appear that if the naso-labial fold has any significance with regard to this lesion, it is more likely to be because of an association with sebaceous glands rather than with embryological cell rests.

Willis is very definite in his opinion on the cell rest theory with regard to basal cell carcinomata. Writing of the latter he makes the following statement:

> On the face the most common sites are on the lower lids, along the side of the nose and across the middle of the cheek. Glasunow and McFarland et al. saw in this distribution a relationship to the embryonic facial clefts and supposed the tumours to be "facial cleft carcinoids" derived from developmentally sequestrated cells. No real evidence exists to support this purely speculative suggestion; indeed, the distribution of tumours is opposed to it, for many quite typical rodent carcinomas arise on parts of the face and neck remote from the facial clefts or in other parts of the body. The predilection of the tumours for the middle third of the face and nasal margins is probably related to the distribution of sebaceous glands and perhaps to the incident of light.

Heredity.

It is well known that some types of skin are more susceptible to actinic cancers than others. Here we have three generations of basal cell carcinomata. The extraction was English-Irish, and the family was of the fair type, though not excessively freckled.

However, the following quotation from Hadfield and Garrod is worth noting.

> The high familial incidence of breast cancer is fully confirmed (112 cases in 301 total cancer cases related to 207 out of a total of 660 original cases of cancer of the breast). On the other hand, a total of 531 individuals with cancer of the skin and lips had only 41 traceable relatives with similar growths . . . the question of whether a hereditary factor plays any part in susceptibility to the action of known carcinogenic agents. The human studies already quoted in so far as they bear on this question answer it in the negative: the influence of heredity is clearly evident only in connection with internal growths whereas epitheliomas of exposed surfaces for which extraneous causes are known sometimes to be responsible. occurs mainly in families with no underlying high cancer evidence.

This, of course, would not include *xerodermia pigmentosum*, in which there appears to be an inherited supersensitivity to light.

Sunlight.

Ultra-violet light, long having been considered carcinogenic, has now been proved experimentally to be so. Hadfield and Garrod state that "excessive exposure to sunlight is fully recognised as a cause of epithelioma", and this "action has been confirmed experimentally by exposing mice to ultra-violet light, which is now even described as a good carcinogenic agent for experimental studies".

The town in which the child in the present case lived is in the dry inland north-west part of Queensland, where there is almost a maximal amount of sunlight. Cloudy conditions are minimal, occurring mostly in the "wet season", which usually lasts for only a few weeks. After the wet season the grass becomes green, but withers after about eight weeks to a natural hay, which is good sheep food but does not abate the glare. The plain over which the girl's house faced was devoid of trees except for an occasional prickly bush.

Dryness.

Barrack considered that the incidence of cutaneous carcinomata was probably relatively higher in the inland areas of Queensland than on the more humid coastal areas.

Other Influences.

Willis, on the subject of actinic cancers, states that "doubtless other carcinogenic factors cooperate with sunlight". Edwards, writing of rodent ulcers, states that "with the exception of prolonged and excessive exposure to the sun, no external agencies appear to influence its development".

Ross, on the subject of skin lesions due to pitch and tar, describes skin lesions of keratoses, papillomata and

epitheliomata, but not rodent ulcers. He also states that "an excess of ultra-violet light accelerates the action of carcinogens in some mysterious way" — epitheliomata appear far more rapidly in rural than in urban districts. However, experimentally Rusch *et alii* showed that there was no summation of carcinogenic effects of hydrocarbons and ultra-violet light.

Lubricating oil as a carcinogenic agent is known (causing mule-spinner's cancer *et cetera*), and petroleum will become carcinogenic if sufficiently heated.

These statements were found while the question was being considered whether contact with an oil depôt could have any influence on the origin of this tumour in so youthful a subject. However, in the case in question the lesion is not of the type (squamous epithelioma) which usually develops from contact with the polycyclic hydrocarbons. The contact would be minimal, especially as the carcinogens are usually in the heavier, less splashable oils, though these would cling more to taps, spanners *et cetera*.

Summary.

The finding of a basal cell carcinoma in a child aged five years led to inquiries which may be of interest to others who meet this condition. Differential diagnosis, age, site, heredity, sunlight, dryness and other carcinogens are considered.

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References.

AREY, L. B. (1937), "Developmental Anatomy", 3rd Edition: 142.

BARRACK, B. B. (1952), M. J. AUSTRALIA, 2: 519.

BOYD, W. (1947), "Textbook of Pathology", 5th Edition: 307.

CLARK, W. E. LE GROS (1946), "Tissues of the Body", 2nd Edition: 276.

CUNNINGHAM, D. J. (1947), "Textbook of Anatomy", 8th Edition: 406.

DARGEON, H. W. (1940), "Cancer in Childhood", St. Louis.

EDWARDS, H. (1948), "Recent Advances in Surgery", 3rd Edition: 423.

FRAZER, J. E. (1946), "The Anatomy of the Human Skeleton", 4th Edition: 222.

FRENCH, H. (1948), "Index of Differential Diagnosis of Main Symptoms", 6th Edition.

GATES, O., WARREN, S., and WARVI, W. N. (1943), "Tumours of Sweat Glands", *Am. J. Path.*, 19: 591.

"Gray's Anatomy: Descriptive and Applied" (1949), edited by JOHNSTON, T. B., and WHILLIS, J., 30th Edition: 543.

HADFIELD, G., and GARROD, L. P. (1948), "Recent Advances in Pathology": 73, 88, 89.

KING, E. S. J. (1948), "Significance of Modern Embryology in Pathology", M. J. AUSTRALIA, 2: 705.

MCGREGOR, A. L. (1946), "Synopsis of Surgical Anatomy": 323.

MOLESWORTH, E. H. (1927), "Rodent Ulcer", *M. J. AUSTRALIA*, 1: 878.

PAUL, N., and INGLIS, K. (1927), "A Case of Epithelioma Adenoides Cysticum (Brooke)", *Brit. J. Dermat.*, 39: 195.

ROSS, P. (1948), "Skin Lesions due to Pitch and Tar", *Brit. M. J.*, 2: 369.

SEQUEIRA, J. H., INGRAM, J. T., and BRAIN, R. J. (1947), "Diseases of the Skin", 61: 4.

TRETHOWAN, H. M. (1952), M. J. AUSTRALIA, 2: 519.

WAKELEY, C., and CHILDS, P. (1949), "Basal Cell Carcinoma", *Brit. M. J.*, 1: 737.

WARVI, W. N., and GATES, O. (1943), "Epithelial Cysts and Cystic Tumours of the Skin", *Am. J. Path.*, 19: 765.

WILLIS, R. A. (1948), "Pathology of Tumours", 272, 285.

WILLIS, R. A. (1950), "Metaplasia", writing in "Studies in Pathology Presented to Peter MacCallum", Melbourne University Press: 53.

A FURTHER CASE OF ACUTE HÆMORRHAGIC LEUCOENCEPHALITIS (HURST).

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HURST first described acute hæmorrhagic leucoencephalitis in 1941, from two cases which had occurred in South Australia. Since then, the total of adequately recorded and acceptable cases has been increased to nine (Hurst, 1941; Henson and Russell, 1942; Shallard and Latham, 1945; Wolf and Cowen, 1946; Adams, Cammermeyer and Denny-Brown, Case IV, 1949; Fowler and French, 1949; and MacArdle, van Bogaert and Lhermitte, 1949). In 1949 Fowler and French reported failure to cultivate virus from the central nervous system of their subject.

All cases of the disease have been fatal, the pathological findings being remarkably uniform. The cause of the disease remains unknown, although there appears to be in some cases an association with respiratory infections. Wolf, Kabat and Bezer (1947) have compared the findings in acute hæmorrhagic leucoencephalitis with the cerebral changes in monkeys following their inoculation with heterologous or homologous brain tissue.

The purpose of the present article is to place on record a further case of this rare disease, in which attempts at virus culture from the central nervous system after autopsy were unsuccessful.

CLINICAL RECORD.

A male patient, aged thirty-three years, was admitted to the Repatriation General Hospital, Springbank, South Australia, in the early hours of the morning of July 29, 1951. He was an officer of the British Army, serving at Woomera, South Australia, his duties normally taking him between Woomera and Adelaide, South Australia, every few days. Normally he enjoyed good health, the only important past medical history being one of head injuries suffered during the Greece and Crete campaigns of World War II, which had affected his vision, though an account of these was not available. He had experienced some headaches during the war, but none recently, until the present illness occurred. In 1945 he was reported to have had malaria and severe jaundice in Germany, but there had been no more recent history of these complaints, or of exposure in a malarious area. There was no history of recent illness in his family or near associates. The only report on his infectious diseases milieu was that there had been a few cases of some mild and undetermined pyrexias at Woomera, thought possibly to be influenza. The onset of his illness was on July 14, 1951, when he complained of a very severe headache—"the worst that he had ever had"—which had come on during the day when he was travelling over some bumpy ground in a jeep. On July 15 he felt ill, but worked hard all day moving house, and fainted during the evening. On July 16 the malaise continued and the headache was still severe. The patient thought he had the "'flu". There was no backache. The headache continued over subsequent days, but in milder form. He continued at work, having always been reticent about complaining. On July 23 his voice became hoarse. On the next day this hoarseness increased and he also complained of a slight soreness in the throat. On July 26 the headache returned in severe form, and there was some vomiting. Diplopia also occurred, and medical attention was sought. The only abnormal physical findings were a temperature of 99·2° F., and some pharyngeal inflammation. Treatment with sulphonamide tablets was instituted. On July 27 he became semicomatose. His headache was less severe. Further examination revealed a reddened pharynx, contracted

pupils reacting to light, and sluggish or absent knee jerks; but the biceps, triceps and ankle jerks were present. The plantar responses were flexor in type. The spleen was not palpable. He was admitted to hospital and given 600,000 units of "Procillin". On July 28 his speech became slurred. He was reported as having had a good night, having vomited only once. A further 600,000 units of "Procillin" were given, as well as 15 grains of quinine. He was unconscious most of this day also, his stupor gradually increasing during the day. He was noticed in the evening to be having difficulty in attempts to clear his throat.

On July 29, at 3 a.m., he was admitted to the Repatriation General Hospital, Springbank. On examination he was semicomatose, unable to answer questions, cyanosed, lying curled up on his left side, breathing quietly. He resisted examination, as well as efforts to turn him on his back. The temperature was 99·6° F. and the pulse rate 104 per minute, and the respirations numbered 28 per minute. The pupils reacted to light. Sordes were present around the mouth. The spleen was not palpable. The liver was palpable one inch below the right costal margin. Examination of the heart revealed no abnormality. The blood pressure was 110 millimetres of mercury, systolic, and 65 millimetres, diastolic. There was slight neck rigidity. The knee, ankle and abdominal reflexes were absent. The plantar responses were extensor in type. Near the dorsal surface of the right wrist was a hairless patch about 3·5 centimetres in diameter. It was stated that this patch had been present for eight weeks prior to the onset of his illness. He was treated with penicillin, 300,000 units being given every four hours. A few hours later the temperature was 97·6° F. and the pulse rate 84 per minute, and the patient lapsed into low muttering delirium. Lumbar puncture was performed. The cerebrospinal fluid pressure was 210 millimetres of water, and the fluid contained 430 cells per cubic millimetre, 69% being neutrophile cells and 31% lymphocytes. In addition, there was a moderate number of smear cells in the cerebrospinal fluid, which appeared to be mainly neutrophile cells. Attempted culture of the fluid produced no growth of microorganisms. The protein content was 145 milligrammes per 100 millilitres, globulin being present in excess ("++"); the chloride content (as sodium chloride) was 750 milligrammes per 100 millilitres; the glucose content was 35 milligrammes per 100 millilitres.

It was considered that the diagnosis was one of encephalitis, or, less likely, some purulent infection of the central nervous system.

In the afternoon the temperature rose to 102° F. The pulse rate was 76 per minute and the respirations numbered 46 per minute. The patient developed right hemiplegia. Aureomycin therapy was instituted, one gramme being given *statim* and then 0·5 gramme every six hours; tube feeding was also begun. Catheterization of the bladder produced a few ounces of residual urine, the patient being incontinent. By evening, further paralysis had occurred, affecting breathing and swallowing. Cyanosis now became pronounced, and the patient was given oxygen continuously and placed in a respirator, after which his colour improved. His respirations remained between 40 and 50 per minute, and he had so much difficulty in accommodating himself to the rhythm of the respirator that eventually he had to be taken out.

On July 30 there was still considerable cerebral irritation. Introduction of the naso-pharyngeal catheter induced coughing. His knee jerks and ankle jerks remained absent, and the plantar responses remained extensor in type. The tonus was equal and normal in both arms. The biceps jerks were present, the right being brisk and the left reduced. The right triceps jerk was present, but the left was not elicited. Tracheotomy was performed in the evening by Dr. R. H. von der Borch, and after that the patient's breathing became easier. Examination of his urine on this day showed it to contain about 10 erythrocytes per high-power field. A blood examination gave the following information: the hæmoglobin value was 12·6 grammes per 100 millilitres; the white cell count was 24,800 per cubic millimetre, 87% being neutrophile cells, 10% lymphocytes, 2% monocytes and 1% eosinophile cells; the neutrophile cells showed a "shift to the right", there being only an occasional juvenile neutrophile cell present; the erythrocytes showed some anisocytosis.

On July 31 the patient's condition was much the same. The limbs were still fairly flaccid, though some tonus was present. He was making mild restless movements of the left arm and leg. The patient remained irritable to the stimulation of handling or examination, and this caused him to grimace his left facial musculature. The *alæ nasi* were working, the left more than the right. Both sides of the diaphragm moved evenly. There was no intercostal retraction on respiration. The knee jerks, ankle jerks and abdominal reflexes remained absent. The biceps, triceps and supinator jerks were present on the left side, absent on the right. The jaw jerk was absent. Cremaster and Chvostek's reflexes were absent. The pupils were large and equal, reacting sluggishly to light. The ophthalmologist (Dr. D. O. Crompton) reported as follows:

> Corneæ a trifle hazy. Disc edges slightly blurred everywhere, except on temporal sides; veins perhaps a little engorged. The appearances on each side are identical, suggestive of slight papillœdema.

The chest skiagram was reported as normal, except for a possible slight opaque area in the right mid-zone, which could possibly have represented consolidation; the film (taken on a portable machine) was considered somewhat unsatisfactory.

On August 1 his condition was unchanged. There was some flickering of the eyelids. The respirations remained at about 45 per minute. The pulse was of good volume and regular, the rate remaining at about 150 per minute. Cyanosis was a little more prominent. Oxygen therapy was continued, and the aureomycin dosage increased to 0·5 gramme every four hours.

On August 2 his condition was deteriorating. His pulse was weaker and rapid. The respirations remained rapid and strong; cyanosis was still present. The gradual deterioration continued, and he died at 11.25 a.m.

Post-Mortem Examination.

Autopsy was performed by Dr. D. L. Wilhelm, three hours after the patient's death, and the following is a summary of his findings.

The right pupil was dilated more than the left. In the lungs there was considerable congestion of the lower lobes with scattered small superficial nodules, resembling broncho-pneumonia. In the liver, stomach, spleen and kidneys mild congestion was present. The brain was found to be generally congested.

Several specimens of brain tissue were taken for virus isolation, and the unsliced brain and cord were then fixed in formol saline and given to us for examination.

Central Nervous System.

Macroscopic Examination.

Examination of the fixed specimen showed that the left cerebral hemisphere was much swollen, with flattening of its convolutions; the superficial vessels all over the brain were very congested. On examination of sections, the left hemisphere was seen to owe its enlargement mainly to changes in the central white matter, the ventricular cavities being reduced to mere slits. The white matter of the *centrum semi-ovale* was affected from a level just in front of the tip of the temporal lobe anteriorly to within one and a half inches of the occipital extremity. The temporal lobe and the insula were also involved. The pre-central gyrus was severely damaged and greatly enlarged, having in the white matter a small cystic cleft-shaped area which measured 2·0 by 2·0 by 0·5 centimetres.

The lesions extended into the internal capsule to a lesser extent, and the subcortical "U" fibres and the optic tracts appeared to be spared. The cerebellum, medulla and mid-brain and spinal cord showed no changes.

The involved areas were soft and hæmorrhagic, the inadequately fixed white matter being discoloured pale yellow with scattered bright-red pin-point hæmorrhages both of the "ring" and "ball" varieties. The softening was so extreme, as mentioned above, as to lead to cystic change in one area. On thorough fixation the affected tissue was of a greyish colour, the immediate subcortical white matter being white. The right hemisphere was normal.

Microscopic Examination.

Celloidin preparations were made from the left lobe of the cerebellum, the *medulla oblongata* and one whole sagittal section from each cerebral hemisphere, on the left side the softened area being included. Pieces from the brain stem and from various levels of the spinal cord, and a specimen of one optic tract were embedded in paraffin. The sections were stained by Weil's myelin sheath method, by van Gieson's preparation and with Nissl's stain.

The most severe lesions were found in the white matter of the left cerebral hemisphere. Naked-eye examination of the sections stained by Weil's technique disclosed great swelling of the white matter, particularly in the pre-central gyrus, where there was a cavity roughly measuring 0·5 centimetre in breadth and 2·0 centimetres long (Figure I). This area was surrounded for a distance of up to 0·5 centimetre by completely demyelinated brain. Another area, approaching cavity formation, was also present in the pre-central gyrus, and here there was almost complete demyelination. The superior temporal gyrus was also severely affected. The small hæmorrhages were largest and of greatest density in these areas. All these affected zones being near the grey matter, the subcortical white matter here showed much demyelination. In the less peripheral parts of the *centrum semi-ovale*, there was a more diffuse appearance of demyelination, probably due to œdema, and complete zones of perivascular demyelination up to a millimetre in radius. Confluence of these latter areas had occurred. The white matter of the gyri was involved, mainly by perivascular demyelination (Figure II), and the subcortical white matter here tended to be spared. The white matter under the insular cortex showed much perivascular and diffuse demyelination and many hæmorrhages. The internal capsule was mildly involved by partial perivascular demyelination.

Under the microscope the edges of the cavity were seen to be composed of granular fragmented brain, in which could be found the pale remains of chromatolytic nuclei, red cells and pyknotic nuclear fragments. Surrounding this area of softening, œdema was pronounced, with diffuse and perivascular demyelination. In areas of partial demyelination the myelin sheaths were irregularly beaded with groups of vacuoles. Hæmorrhages were most frequent and severe in and around this area. The areas surrounding blood vessels were infiltrated by scavenger cells (Figure III) and a few polymorphonuclear cells amongst pyknotic glial nuclei. Polymorphonuclear cells were sometimes numerous in the walls of venules and in the immediately surrounding brain; but only here were they well preserved, for most at any distance from a vessel showed karyorrhexis or pyknosis.

In the right cerebral hemisphere, lesions were confined to the *corpus callosum* and the white matter of the *gyrus cinguli*, and were all perivascular. Polymorphonuclear cells were conspicuous around some vessels, and there was active proliferation of microglia, most being in the rod-cell stage with incompletely mobilized cytoplasm. Mitotic figures were numerous amongst them. An occasional member with foamy well-delineated cytoplasm was seen. Perivascular demyelination was moderate.

In the central white matter of the cerebellar hemisphere there was much perivascular demyelination, but the white matter of the folia was unaffected. The main reactive cells were microglial, with vacuolated cytoplasm, and only after a determined search was a rare polymorphonuclear leucocyte found. Hæmorrhages were very uncommon. Nerve cells were not involved, except for one area in the dentate nucleus abutting on an area of demyelination, and here there was some tigrolysis and neuronal swelling without neuronophagia. Patchy mild lymphocytic infiltration had occurred around some of the large meningeal vessels. Apart from an occasional tiny hæmorrhage and sparse meningeal infiltration with lymphocytes, the spinal cord, medulla and optic tract were not abnormal. The choroid plexus was normal.

Other Tissues.—The lungs were œdematous, the fluid often containing many red blood cells. A few small round cells were occasionally seen in the wall of a bronchiole, but there was no other cellular infiltration. Examination of sections of the cervical lymph glands revealed congestion, a little sinus hyperplasia, indefinite lymphoid follicles without germinal centres, and the slight generalized presence of atypical plasma cells and some young neutrophile polymorphonuclear leucocytes.

Virus Investigations.

Parts of the cortex, basal ganglia, cerebellum, pons, medulla and cord were pooled, ground with silica and extracted as a 20% weight for volume suspension in 10% rabbit serum and saline. Five hundred units per ml of penicillin and streptomycin were added to the inoculum before injection.

The material was passed to fifth-day eggs by yolk sac inoculation and to twelfth-day eggs by chorio-allantoic inoculation, the material being used as 20%, 2% and 0·2% suspension. Chorio-allantoic eggs were harvested after seventy-two hours, and yolk sac eggs after seven days. Material from eggs inoculated with all dilutions of original brain extract was pooled and passed as 20% suspension for two further blind passages. Since neither macroscopic nor microscopic findings gave evidence of infection, the passage was then abandoned.

The same material was passed at 20% and 2% intracerebrally to three to four weeks old weaned mice and given three blind passages without any evidence of infection. A single intracerebral passage in suckling mice, rabbits and guinea-pigs also gave negative results.

After the material had been stored for six months in dry ice, until monkeys became available, two young rhesus monkeys were inoculated intracerebrally and intranasally with 0·5 millilitre of 20% suspension. Neither of these monkeys showed any pyrexia during one month after inoculation, or other evidence of ill health during three months after inoculation.

Bacteriological investigations were not made.

Discussion.

The main point worthy of note in this case is the relatively long period of illness of eighteen days. In the previous reports, the patients have not lived longer than six days from the onset of cerebral symptoms. Our patient suffered from a relatively long period of premonitory headache and malaise, followed by survival of seven days after the first localizing neurological sign (diplopia) appeared.

The pathological findings differ slightly from those of the previously reported cases, slides from three of which one of us has examined. Polymorphonuclear leucocytes were far more conspicuous than in the present case, in which they were relatively scanty. Recent perivascular necroses have usually been found; none was discovered in our case—because of either lessened severity of the destructive process or the removal of the necrotic material by the microglial activity which was much in evidence. However, hæmorrhages of very recent occurrence were found, but no evidence of hæmorrhages likely to have occurred earlier in the illness.

The presence of a liquefied area of such extent has not previously been observed in this condition, and this is most reasonably attributed to the long survival time in our case. Hurst (1941) mentions an area "wholly gelatinous" in his first case.

Again, the unusual unilaterality of the process in the cerebral hemispheres is observed, almost completely in this case except for small early lesions in the vicinity of the *corpus callosum* on the right side. This distribution is unlike virus infection, and on this occasion, and once previously (Fowler and French, 1949), attempts to isolate an infectious agent have failed. The manner of action of the harmful agent is peculiar, in that the two cerebral hemispheres apparently similar in blood supply and other anatomical constitution are so unequally affected. If the process is one mediated by the blood-stream, the unilaterality of the lesions must be due to differing susceptibility of the two hemispheres, differing susceptibility being conditioned by difference in functional activity.

An alternative hypothesis is that the entrance to the nervous system is gained from a unilateral source and the infection ascends axonally to the opposite hemisphere.

A primary disorder of blood vessels is unlikely, for the grey matter is only secondarily affected from changes in the adjacent white matter, and it also does not explain the unequal changes in the two hemispheres.

Of the ten cases, including the present one, in four the lesions have been confined to the left hemisphere, in two predominantly on the right side, in two predominantly on the left, and in one case the lesions were equally distributed. (The account of Wolf and Cowen's case is not available in Australia.) The unilaterality of the process is therefore manifest; but whether the predominance of involvement of the left hemisphere is significant may be decided when more cases are published.

The lesions in the lungs in our case are those secondary to the comatose state, and other organs offered no evidence of infection.

SUMMARY.

A further case of acute hæmorrhagic leucoencephalitis is described, in which the duration of the illness was eighteen days, the longest recorded in this disease.

The pathological investigations produced evidence of longer duration of the process than in previous cases, and emphasized the unilateral character of the disease.

Virus investigations were fruitless.

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REFERENCES.

ADAMS, R. D., CAMMERMAYER, J., and DENNY-BROWN, D. (1949), "Acute Necrotizing Hemorrhagic Encephalopathy", *J. Neuropath. & Exper. Neurol.*, 8: 1.

FOWLER, M., and FRENCH, E. L. (1949), "A Case of Acute Hæmorrhagic Leucoencephalitis (Hurst) Following Atypical Pneumonia, with a Description of Cultural and Virus Studies", M. J. AUSTRALIA, 1: 267.

HENSON, R. A., and RUSSELL, D. S. (1942), "Acute Hæmorrhagic Leuco-encephalitis: Report of a Case", *J. Path. & Bact.*, 54: 227.

HURST, E. W. (1941), "Acute Hæmorrhagic Leucoencephalitis: A Previously Undefined Entity", M. J. AUSTRALIA, 2: 1.

MACARDLE, J., VAN BOGAERT, L., and LHERMITTE, F. (1949), "*La leuco-encéphalite hémorrhagique aiguë (Hurst)*", *Rev. neurol.*, 81: 709.

SHALLARD, B., and LATHAM, O. (1945), "A Case of Acute Hæmorrhagic Leucoencephalitis", M. J. AUSTRALIA, 1: 145.

WOLF, A., and COWEN, D. (1946), "Transactions of the American Association of Neuropathologists", June 26; cited by Wolf, A., Kabat, E. A., and Bezer, A. E., *loco citato*.

WOLF, A., KABAT, E. A., and BEZER, A. E. (1947), "The Pathology of Acute Disseminated Encephalomyelitis Produced Experimentally in the Rhesus Monkey and Its Resemblance to Human Demyelinating Disease", *J. Neuropath. & Exper. Neurol.*, 6: 333.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Current Therapy, 1954: Latest Approved Methods of Treatment for the Practising Physician", edited by Howard F. Conn, M.D.; 1954. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 10½" × 8", pp. 930. Price: £5 4s. 6d.

No less than twelve consulting editors have had a hand in the production of this book; there are 192 contributors.

"Mayo Clinic Diet Manual", by The Committee on Dietetics of the Mayo Clinic; Second Edition; 1954. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 9½" × 6½", pp. 260. Price: £2 12s. 3d.

A practical manual setting out the diets for use in a great variety of conditions. There is a useful index.

"Methods in Medical Research", Editor-in-Chief, J. Murray Steele; 1954. Chicago: The Year Book Publishers, Incorporated. Volume VI. 8½" × 5½", pp. 284, with 49 text figures. Price: $7.00.

Deals with human genetics, environmental research, statistics in medical research and the design and construction of metabolism cages.

"Practical Electrocardiography", by Henry J. L. Marriott, M.D.; 1954. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6", pp. 182, with 115 illustrations. Price: 53s. 9d.

Designed for those approaching electrocardiography from the point of view of the clinician.

"Clinical Neurology", by Bernard J. Alpers, M.D., Sc.D. (Med.); Third Edition; 1954. Philadelphia: F. A. Davis Company. Sydney: Angus and Robertson, Limited. 10" × 7½", pp. 896, with 243 illustrations. Price: £5 2s. 3d.

An attempt to make the subject of neurology "intelligible" to medical students and general practitioners.

"Diseases of the Knee: Management in Medicine and Surgery", by Anthony F. DePalma, M.D.; 1954. Philadelphia: J. B. Lippincott Company. Sydney: Angus and Robertson, Limited. 10½" × 7½", pp. 862, with 455 illustrations. Price: £10 15s.

Intended not only for the orthopædic expert, but also for the general surgeon, the general practitioner and the student.

"Clinical Interpretation of Laboratory Tests", by Raymond H. Goodale, M.D.; Third Edition; 1954. Philadelphia: F. A. Davis Company. Sydney: Angus and Robertson, Limited. 9" × 6½", pp. 738, with 105 illustrations, six in colour. Price: 80s. 9d.

First published in 1949.

"Almroth Wright: Provocative Doctor and Thinker", by Leonard Colebrook, F.R.S.; 1954. London: William Heinemann (Medical Books), Limited. 9" × 5½", pp. 298, with 28 illustrations. Price: 21s.

The author has tried to convey something of Wright's personality against the background of his adventures in medical science.

"Gehirn und Auge: Grundriss der Ophthalmo-Neurologie", by Robert Bing and Roland Brückner; Third Edition; 1954. Basel: Benno Schwabe and Company. 9½" × 7", pp. 248, with 147 illustrations. Price: 28 francs (Swiss).

One author is professor of neurology and the other professor of ophthalmology at the University of Basel.

"Practical Obstetrics", by Bruce T. Mayes, M.V.O., M.B., B.S. (Sydney), F.R.C.S. (Edinburgh), F.R.A.C.S., F.R.C.O.G., with a foreword by John A. Stallworthy, M.A. (Oxon.), F.R.C.S. (England), F.R.C.O.G.; Second Edition; 1954. Sydney: Angus and Robertson, Limited. 9" × 6", pp. 514, with 181 illustrations, 21 in colour. Price: 87s. 6d.

First published in 1947; this is the second edition.

The Medical Journal of Australia

SATURDAY, JULY 10, 1954.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE JOURNAL'S FORTIETH BIRTHDAY.

On July 4, 1954, THE MEDICAL JOURNAL OF AUSTRALIA celebrated its fortieth birthday—the first issue having seen the light on July 4, 1914, just one month before the outbreak of the First World War. The younger members of the medical profession in Australia need perhaps to be reminded of the fact that the establishment of this journal was the outward and visible sign of the union which had taken place between the six Branches of the British Medical Association in Australia by the establishment of their Federal Committee. The first article in the first issue was entitled "Notes on Cardiac Irregularities" and was written by Dr. M. D. Silberberg, of Melbourne, who has taken a practical interest in the affairs of the journal ever since that time and is still practising in Melbourne. This number also contained an article by Dr. J. H. L. Cumpston in which the history of medical journalism in Australia was reviewed. That article has been used as a standard of reference ever since it appeared. The journal appeared every week and was produced under the editorship of the late Henry William Armit. The first volume contained 616 pages. In those days the original articles were printed in what is known as ten-point type, and eight-point was used for the part of the journal which followed the leading articles. Today, the journal is printed in eight-point and seven-point, which means that much more material can be produced in one page. The half-year which has just been completed contained 1000 pages of reading matter. Since THE MEDICAL JOURNAL OF AUSTRALIA was established, we may claim that it has taken a prominent part in the affairs of the medical profession of this country, and that it has catered for practitioners of all kinds. The old saw has it that life begins at forty. If that is so, we may claim to have had an eventful and vigorous adolescence and we are determined, with the help of the members of the medical profession (it cannot be done without them), that our future life will be vigorous and more fruitful.

GENTLENESS AND THE APPROACH TO THE PATIENT.

LIKE love, joy, peace, long-suffering and the others, gentleness is one of the "fruits of the spirit". It is a quality most to be admired among men, whether they are men of fame or fortune, of high degree or low; it is a quality indispensable to the "good doctor". In this phrase the word "good" refers not so much to the doctor's moral worth, important though that may be, as to his capacity to understand and be understood by his patients and his ability to relieve their sufferings of body and mind. The capacity to be gentle is not always understood. The word is of ancient usage in the sense that a man has been described as a gentleman or as not a gentleman. We do not need to be reminded that the title has savoured of snobbery—the "Gentlemen" oppose the "Players" at cricket and so on. A man born in a certain social circle was said to be a gentleman; he might have been a rough, boorish, and intolerable person, but his status of birth carried the implication of gentility. We know, of course, that a man in any social stratum may be a gentleman—that is, he has thoughtfulness for others and does not push himself to the front without regard to his neighbour. Cardinal Newman knew this when he described a gentleman as one who never inflicted pain. We recall, too, a certain novelist who is said to have referred to a well-known actor as being "every other inch a gentleman". There are many such people in the world—sometimes the proportion is much less than every other inch. The polish that comes from the cultivation of good manners must not be confused with the quality of gentleness. We can probably all recall the smooth rogue whose suavity is but a veneer to his hardness of heart and cruelty of action—he can "smile and smile and be a villain". Gentleness is not necessarily weakness, and we know that weakness may easily turn into hardness. Someone has said that power can do by gentleness what violence fails to accomplish. The Duke in *As You Like It*, in the scene with Orlando and Jaques, remarks:

> ". . . your gentleness shall force
> More than your force moves us to gentleness."

The medical practitioner, in his dealings with patients, remembers, or should remember, always the injunction: "*Primum non nocere*"—above all things, to do the patient no harm. We may add to this that the practitioner's every word and action should be gentle. The approach to the patient by the medical attendant must be made in such a way that the patient loses any apprehension or fear that he may have had of the doctor as a person, or of the possible ordeal that the doctor may wish to impose upon him. This is a question of faith which has been discussed from this point of view in these pages on a previous occasion. The approach of the doctor to his patient will depend on the approach of the latter to the former. The approach is mutual. The doctor it is who has to determine a course of action, and his bearing will depend in some degree on the attitude of the patient. The apprehensive

and agitated patient has to be treated not very differently from the way in which the dejected or fatalistic sufferer seeking advice is treated. The patient must always see before him a calm, self-reliant and reliable individual; thus alone is trust engendered. If to these qualities are added gentleness of word and action, little more will be needed. This discussion might be prolonged by reference to the art of diagnosis and what the patient should be told, and to the difficulties of prognosis and the wisdom of attempting to forecast the future in an illness. But this is really not necessary. C. I. Hagger, in addressing the Guy's Hospital Dental Society last October, told his audience[1] that they would need the tact of a diplomat, the sagacity of a saint, and the patience of the proverbial patriarch. This is in large measure true in medicine. Many will acquire tact, sagacity and patience in greater or lesser degree; if gentleness goes with these other qualities, not much that is good will in the long run be lacking. The doctor will be a gentleman and also a gentle man. In conclusion, we may contemplate one of the Beatitudes, which states that: "Blessed are the meek for they shall inherit the earth." This was translated by E. V. Rieu, in his book "The Four Gospels", as follows: "Happy the gentle, for they shall inherit the earth."

Current Comment.

AN EXPERIMENT WITH PEPTIC ULCER.

IN 1943, when the Royal Navy was acutely aware of the need to retain skilled men in the Service, the Admiralty sanctioned the provision of special dietetic arrangements and conditions of service at certain naval bases to which suitable men with proved peptic ulcer were drafted. The scheme, which is described by Charles Newman and his collaborators in Chapter II of the first volume of the United Kingdom Official Medical History of the Second World War ("Medicine and Pathology"), is stated never to have attained more than experimental status, but a few points of interest emerge from the experience gained. Some 70 men in all were selected and employed on harbour and dockyard duties, as engine room artificers, electricians, seamen, stokers, signal ratings and cooks. They were divided into two groups, each being a corporate unit with its own messing arrangements, and were subject to all the rules of naval life except that, firstly, they were static and were excused work involving special hardship or fatigue, and secondly, they had regular meals and snacks between meals and extras in the way of milk and eggs. Only men of a special value to the Service and of good morale were picked. This, it is considered, probably accounts for the fact that though they all had unequivocal evidence of peptic ulcer at selection and were employed for a year, the percentage of their total working days spent off duty sick with dyspepsia was only 8·78. Nevertheless, while 73% of the men under these favourable conditions were sick only for a week or less, 14% were off sick for appreciable periods, which made their retention uneconomic. The experiment concluded with the end of hostilities with Germany. Only 21 out of the 70 men were free of symptoms and showed no radiological evidence of gastro-duodenal abnormality. Twenty-five men had both symptoms and radiological evidence of abnormality. When these were contrasted with the others in whom improvement or recovery had been effected, little significance seemed to attach to the previous duration of symptoms, occurrence of complications or age. In the chronic active ulcer group, however, a high proportion were of unstable temperament, lacking in cooperation or insight, and immoderate in their habits in regard to the use of tobacco and alcohol. Psychogenic factors as such appeared to play little or no part, and had, in fact, been largely excluded.

That is how the matter stood in 1945, but James W. Rae and R. S. Allison,[1] who were concerned with the original experiment, have now reported a follow-up study of most of the patients made at least five years after the end of the original experiment. From their findings they attempt to throw some light on a number of questions relating to the natural history of peptic ulcer. The follow-up investigation was conducted by a postal *questionnaire*, to which 52 men out of 63 replied. Of the 52, only four had been completely free from dyspepsia during the previous five years, five had experienced symptoms only on very rare occasions, and 43 had had recurrent symptoms which were severe enough to interfere with their working capacity; 32 had been off work for more than a week every year on account of dyspepsia, and ten had lost more than three weeks a year. Four had suffered complications—melæna, perforation or pyloric stenosis. Over two-fifths of the patients who were traced still followed a fairly regular treatment regime of diet and alkaline powders, while one-fifth had given up all forms of treatment. The remaining two-fifths took alkalis, combined with diet in about half the cases, only when symptoms recurred. Most of the men who still followed a fairly regular regime belonged to the groups in which periodic symptoms had continued. With regard to the recurrence of symptoms, half of the men related this to worry, but many also remarked that irregular meals or carelessness with diet, tobacco or alcohol might aggravate the dyspepsia; the remainder stated that the attacks appeared to occur without any obvious precipitating cause. Further analysis of the findings with regard to anxiety suggests that this, when it is a prominent feature, may be regarded as a personality trait rather than a specific external influence. The information obtained about tobacco and alcohol makes it doubtful whether these factors can have had much influence on the natural history of the condition.

In a general discussion of the findings, it is pointed out that the majority of the patients dieted or took alkalis regularly only when their symptoms recurred—a finding in line with the view that the natural course of peptic ulcer is the main factor which determines whether the patient will persevere with treatment. Rae and Allison state that when it is remembered that the patients had lived under a common regime for at least a year while under observation, and that they had a better understanding of their condition than the average hospital patient, the findings serve to support the conclusion that the natural history of the disease is not influenced to any appreciable extent by such a lengthy period of treatment and rehabilitation. Moreover, the natural history of the disease appears to be responsible for a formidable rate of recurrence even under the most ideal conditions of after-care. Various investigators have strongly challenged the orthodox methods of management of peptic ulcer, and in particular the dietetic treatment, and the findings in this study would appear to support such challenges. Rae and Allison also question the wisdom of handing out standardized instructions to patients, since every patient presents an individual problem; and whatever may be understood by the term "ulcer diathesis", it must be recognized that the patient should be studied and treated as a whole, his ulcer being regarded merely as an incident in the natural history of the disease. They quote the statement of Jordan that "the life history of ulcer can be said to end only with the life of the patient". They also refer to an editorial statement in the *British Medical Journal* in 1943, to the effect that to separate the ulcer patient from his diathesis was like severing the fisherman from his soul, and until some new secret of Nature was learnt, it was necessary to be content to try to teach the patient how best to live at peace with his ulcer—and to do that he must probably learn how to live at peace with himself. Rae and Allison comment that if the world as a whole was to attain that ideal state, peptic ulcer might no longer be a problem to the medical profession. They conclude with

[1] *Guy's Hosp. Gaz.*, March 6, 1954.

[1] *Quart. J. Med.*, October, 1953.

the rather wry statement that in these days of changing fashions in medicine and surgery, it is perhaps too early to say whether the separation of the patient from his stomach can offer more than a temporary solution to the problem.

THE PREVENTION OF MENTAL ILLNESS.

THE maintenance of huge and ever-increasing mental hospitals constitutes an economic embarrassment to many countries, and, although there must be no question of economizing at the expense of the mentally ill in the community, any sound scheme to reduce the number of those requiring treatment in mental hospitals should be considered. An obvious approach is, of course, the prevention of mental illness, and T. M. Ling[1] has recently drawn attention to some of the methods being used in Holland. Ling writes from the background of the present position of overcrowding in Britain. He points out that Holland has a congested urban and industrial civilization and that its social problems have many points in common with those of Britain, but it is broadly true that during the last twenty years the Dutch have developed a preventive mental health programme planned to keep people, as far as possible, out of hospital, while the British have succeeded in overfilling their mental hospitals. In this respect, Australians seem to have been sticking firmly to their British heritage. Ling states that the Dutch are realistic in their approach and are conscious of the economic implications of social and health services and conditions. In Amsterdam, a city of 850,000 people, the mental health service is run by the city authorities from a central building. A twenty-four hours' service is provided by a team of psychiatrists and social workers, who may be called in by the family doctor, by officials of the Ministry of Labour, or by the police. Once a patient comes under their care, psycho-social treatment is provided in the home, if necessary for months or years. The Dutch believe that mental illness is often caused by a breakdown of social relationships in the family or at work, and that it should be treated in close relation to the family and work situation. In Amsterdam, patients are sent to the hospital only as a last resort, and this step is regarded as an admission of failure by the mental health service. The mental health service is closely concerned with factory and labour conditions and believes that work-placement, aided by regular home supervision, is often the best way of retaining patients as productive members of society. It also runs four sheltered workshops, where patients can be reconditioned for work. Great stress is placed on the home as the basis of the normal family, and on the view that only in the home is it possible to cooperate with the family. An excellent relationship has been built up with the police, who are encouraged to report significant anti-social behaviour to the service. This procedure, it is stated, is often more conducive to a permanent solution than a prosecution in the courts. The service also advises the judiciary in more serious charges. The work of the service in Amsterdam is carried out in teams; each district has a full-time psychiatrist, who is helped by social workers. Although much of their work is done in the home or factory medical department, the doctors of the service have access to first class diagnostic facilities for psychological and laboratory tests. Close liaison is maintained between the service and the university psychiatric clinic, which has 200 beds and is an active teaching and research centre. Ling states that about 40% of all adult psychiatric patients referred to the mental health service are never admitted to hospital, and respond with varying success to handling in the home and factory. When institutional treatment is essential, this is arranged; and on the patient's discharge from hospital the service resumes supervision and attempts resettlement at work. Ling comments that comparisons between one country and another are notoriously difficult and one cannot readily judge whether Amsterdam, which has been developing a first class mental health service for twenty years, is mentally healthier than London, but the suicide rates and the number of people admitted to mental hospitals would both appear to favour Holland. In any case, as Ling points out, there are better ways of spending public money than building more mental hospitals, and the Dutch experience deserves careful study and adaptation to local needs if it appears to be likely to be of value.

[1] *Lancet*, May 29, 1954.

THE DIETARY MANAGEMENT OF PEPTIC ULCER AND ULCERATIVE COLITIS.

To most physicians the medical therapy of gastric ulcer is largely a matter of diet. The aim of treatment is to inhibit, neutralize or dilute the excessive secretion of corrosive gastric juice. The most physiological way of inhibiting gastric function is by the ingestion of a fat-containing meal. Stewart Wolf has given a critical appraisal of the effects of diet in the treatment of peptic ulcer and ulcerative colitis.[1] A subject, Tom, with a gastric fistula was used in the experiments on the stomach. With a normal fat-containing breakfast, the composition of which is not given, at the end of 5·5 hours about 25% of the meal still remained in the stomach, and the free acid content was very low. When fat was omitted from the breakfast, at the end of 4·75 hours the stomach was nearly empty of food and there were about 85 units of titratable acid. When the fat-containing breakfast was consumed in a setting of anxiety and tension the usual effect of fat was entirely outweighed by the stimulating effect of the situation. Only five hours after a meal the stomach was completely empty of recognizable food. Titratable acidity was 100 units and motor activity was unusually vigorous. One then cannot count very heavily on the inhibitory effect of diet in peptic ulcer, especially when the stomach is under stimulation from meaningful events in the individual's daily life. There is little evidence that the wrong food can accelerate gastric function, although coffee and alcohol promote increased gastric acidity and might profitably be eliminated from the diet. The stimulating effects of food are transitory and not very vigorous in comparison with the stimulating effects of life situations. Much has been made of the notion that certain articles of diet are irritating to the stomach. Observations on Tom showed no food, including chilli con carne, irritating to the stomach. The observations were repeated on another fistulous subject with a duodenal ulcer with no evidence of irritation. The skin of the forearm was found to be far more vulnerable to irritants than the lining of the stomach. Thus mustard (1 in 30 suspension in water) produced only a slight erythema in the stomach, but vesiculation and destruction of tissue on the forearm. As far as diet is concerned, it is evidently important to provide feedings that are frequent enough so that the stomach will not remain empty, and it is also important that these feedings contain milk and cream, which do as well at neutralizing and inhibiting gastric function as can be done. The addition of non-absorbable alkalis and antispasmodics is probably of little value. The most important steps in management of patients with peptic ulcer are concerned with a consideration of the person in the patient including the problems of everyday life.

The treatment of ulcerative colitis is also concerned with dietary rituals for which there is little concrete evidence. The fundamental disturbance in ulcerative colitis is one of colonic hyperfunction with hyperæmia, engorgement and intense hypermobility of the gut, with fragility of the membrane and easy ulceration. Ulceration has been produced in a segment of the gut isolated from the fæcal stream and free from all local irritation. The ulcerations occurred in a setting of intense emotional conflict. The gastro-colic reflex is greatly exaggerated in ulcerative colitis, even when the colon has been disconnected from the rest of the alimentary tract. It therefore appears that the ingredients of the fæces, with respect to residual food constituents, have very little to do with colonic hyper-

[1] *J. Clin. Nutrition*, January-February, 1954.

function or ulceration. Patients with no dietary restrictions, but with major consideration of the life adjustments, did better over a three-year follow-up period than a similar group treated by traditional methods including dietary restriction. Undue regimentation or restriction may lead to emotional disturbances with deleterious effects which far outweigh the beneficial effects of the diet. On the other hand a carefully balanced diet, with a certain amount of ritual, may have a very salutary psychological effect on an over-meticulous patient.

GROWTH OF THE HAIR.

THERE are many misconceptions about the growth of hair and the loss of hair. Although the general features of hair growth have been known for many years, the precise details have only recently come under serious investigation.

H. B. Chase has given a detailed review of the present state of knowledge about hair growth and the factors which may affect it.[1] Hair growth is cyclic as regards the particular follicle which produces the hair. The hair increases in length during the "growing" phase of the follicle, then it is retained as a dead club hair during the subsequent "resting" phase. With the next "growing" phase, the old club hair may be lost. In man, as contrasted with most animals, there is neither seasonal growth of hair nor a general wave of growth, and club hairs are not retained after the new hair starts growing. Each follicle has its own cycle and is independent of neighbouring follicles. A follicle in the scalp of man may produce hair for two to six years and then there will be a resting period of at most a few months.

Hair follicles form as solid cylindrical downgrowths from the basal layer of the epidermis. The lower tip of a cord becomes dimpled and the dimple encloses cells from the dermis which become the dermal papilla, connecting the hair follicle with the underlying connective tissue. The cells surrounding the dermal papilla show high mitotic activity and form the bulb. From this cells are pushed upwards and become keratinized. Pigmentary dendritic cells or melanocytes become aligned along the papilla cavity and each develops melanin granules. When new hairs are growing from the follicles the internal sheath grows up as an extended cone to the club of the old hair. New hair is being formed, but is still enclosed by the cone of the internal sheath. In the upper part of the bulb a cone of cells, which will become cortex and medulla of the hair, contains pigment granules. That the presence of dendritic pigment cells is essential for pigmentation of the hair has been fully demonstrated, but the method of transfer of melanin particles is not known. As the region of the hair shaft is reached melanin granules are found mostly within the cortex and medulla cells, and few or none between the cells. The tip of the hair breaks through the internal sheath and grows up to the level of the epidermis and the bulb attains its characteristic shape. In the next stage the hair emerges beyond the surface of the skin and, in man, may continue to grow for two or more years at the rate of about one-half millimetre per day.

The next stage is the resting period. Mitotic activity ceases, the club and capsule of the resting hair are formed and the bulb and lower external sheath are reduced to an epithelial strand of cells the majority of which degenerate. After a varying period of time the whole process starts again and the club hair is shed. Plucking of club hairs provides a stimulus which causes the resting follicles in only the plucked area to start the growth process. Clipping of hair does not initiate growth. Chemical irritants also initiate growth of follicles. Certain doses of X irradiation can destroy pigment cells, resulting in the growth of white and partly pigmented hairs. The pigment cells are more completely destroyed when a resting follicle is irradiated. The white hairs occur only with the next and all subsequent hair generations. Moderate radiation (300*r* to 1000*r*) affects the resting follicle only little apart from the pigment cells. The club hair remains in the follicle and there is only a slight delay, if any, in starting new growth. In the case of the growing follicle, however, the hair falls out in a few days after irradiation. The follicle degenerates, but not completely, and a new hair cycle commences. As long as some potential pigment cells are left the new hair will be pigmented. Larger doses may cause the resting club hairs to fall out, but the follicle is not usually seriously affected.

Methylcholanthrene applied to the skin can cause the hairs to drop out, but new hairs form. There is evidence that the growth of hair and the maintenance of the skin are closely related. Mitotic activity and consequently the thickness of the epidermis increase in the early stages of the growth of a new hair, but by the time the hairs are being produced this epidermal mitotic activity is reduced and is restricted, almost entirely, to the lower part of the bulb of the hair follicle. Glycogen is found in the fibroblasts of the connective tissue sheath; it is most abundant in the cells of the external sheath, especially the middle portion, in active follicles. The cells in the cuticle of the cortex and those which comprise the medulla are rich in glycogen before they become keratinized. The glycogen acts as a readily available source of energy and is therefore not susceptible to the fluctuations in carbohydrate supply which give the diurnal variations found in the basal layer of the epidermis. The resting follicles show little glycogen. What controls the hair cycle is quite unknown.

[1] *Physiol. Rev.*, January, 1954.

PRESSURE AND THE BED-SORE PROBLEM.

BED-SORES can mostly be prevented by efficient nursing, but sometimes their behaviour is puzzling. Their pathology is apparently somewhat more complex than has at times been appreciated in the past. Various investigators have played a part in piecing together the picture, and a particularly useful contribution has been made recently by T. Husain,[1] who has correlated his findings on the experimental effect of pressure on tissues with the previously reported findings of other investigators. He defines bed-sores as foci of skin and subcutaneous tissue necrosis most often found in dependent portions of the body when the soft parts are subjected to long-continued pressure between the bed and some more or less superficial bone. The common sites, as is well known, are the sacrum, greater trochanters, scapulæ and spinous processes of the vertebræ. More rarely, when the tissues become very susceptible to trauma or infection, bed-sores may appear on any part of the body even when the pressure lasts for a short time or is slight, such as that produced by the mere weight of the bedclothes or of a hand lying on the abdomen. Even the resting of one cutaneous surface against another, as with the vulva or scrotum, may suffice to bring about tissue death in special circumstances, especially if there is contamination by fæces or urine.

Clinical experience has made it clear that the production of bed-sores is associated with certain aspects of the patient's general condition, with the condition of his nervous system, with the presence of skin contaminants, such as fæces or urine, and with localized pressure. General debilitating conditions are important as predisposing factors. These, Husain points out, result in nutritive disturbances and anæmia, which may well upset the resisting properties of the skin and muscle. Certain lesions of the central nervous system have been associated with trophic disturbances, including bed-sores. General agreement has not been reached on just what happens in the presence of nervous injuries, but Husain says that the year-long argument about whether special trophic nerves are implicated, or whether vasomotor disturbances alone are responsible for encouraging the formation of bed-sores, seems to have been settled for the time being in favour of the latter view. The role of contamination by fæces or urine in providing mechanical and infective aggravating

[1] *J. Path. & Bact.*, October, 1953.

factors needs no elaboration. The role of pressure is the one to which greatest interest is attached.

Some years ago, Hugh C. Trumble, writing in this journal,[1] discussed the reaction of the skin to pressure influences. He stated that two factors, the intensity and the duration of the pressure influence, must be considered; but he made it clear that of these duration is the more important. Husain's conclusions are in accord with this, and he points out that low pressure maintained for long periods of time produces more tissue damage than high pressure for short periods. Perhaps even more important is the fact that pressure evenly distributed over a wide area of the body is much less damaging to the tissues than localized or point pressure. Husain describes the ways in which point pressure leads to tissue injury. The first is by increasing the permeability of the capillaries in the compressed regions when a certain threshold is exceeded, and especially after release of compression; interstitial œdema develops, lymphatics and venous channels become choked up, and cellular damage may follow. The second is by slowing the blood flow to the part with production of ischæmia and its after-effects. The third is by predisposing to the localization of bacteria at the compressed sites, if bacteriæmia is present before or during compression. Release of persistent compression is followed by reactive hyperæmia, œdema, and often capillary and venous hæmorrhage in the affected region. This stage favours the localization of infection in the area involved and may embarrass the circulation still more, so as to precipitate ischæmia. Amongst factors in the reduction of the threshold pressure level Husain includes partial interruption of the arterial supply to the part (as may occur with obstruction of the femoral artery), division of the main nerve to the part, spinal cord transection and a general constitutional upset, such as vitamin *C* deficiency. In such circumstances, Husain states, mild localized compression may cause serious damage, especially to muscle.

Pathological changes induced by effective point pressure, that is, pressure which has exceeded the threshold level, include loss of muscle striation, conversion of sarcoplasm into homogeneous material, and fragmentation, granularity and eventual necrosis of the muscle fibres. Absorption of the necrosed part of the muscle fibres leads to the production of empty sarcolemmal husks. Pressure not only changes the morphology of the muscle, but also impairs its functional capacity, and this, in turn, may interfere with its blood supply. Husain goes on to point out that histological evidence of muscle damage can be demonstrated in the tissues deep to human bed-sores. This appears to be the result of prolonged pressure rather than infection, and almost certainly precedes the bed-sores. It depends upon anatomical relations, such as nearness to the bony tuberosities and protuberances, and is especially likely to occur when the subject is suffering from conditions which lower the pressure threshold.

This association of bed-sores with initial deep lesions within the muscles is a modern theory based on evidence put forward a few years ago by K. E. Groth. The main sources of evidence are two: firstly, pathological studies which have demonstrated muscular lesions long before superficial bed-sores appear, or have shown that muscle lesions, recent or old, very often accompany bed-sores; secondly, experimental investigations into the mode of action of pressure, which have shown the susceptibility of muscle to physical disturbances as contrasted with the relative resistance of skin and to a lesser extent of fat. Husain's observations agree with those of Groth, and, in addition, he suggests that movement may be of some importance in preventing bed-sore development, for he has found that compressed muscle loses something of its power of contraction in response to stimulation. This, no doubt, adds to the likelihood of prolonged ischæmia from pressure. Husain points out that a flaccid, atonic and feebly contracting muscle is the site of diminished blood flow and that this in turn favours thrombosis and the localization of bacterial infection in its substance. He stresses the need of an adequate supply of vitamin *C* if severe lesions are to be prevented at the site of prolonged compression. He has also found some support for the view that nervous disturbances favour the development of bed-sores, and lastly, the reduction of arterial blood supply to a part makes that part vulnerable to mild pressure, even though the vascular disturbance alone is without ill effects. As he points out, this may have an application in human pathology in relation to the suggestion that arteriosclerotic thickening of vessels predisposes an aging, bedridden patient to bed-sores.

[1] M. J. AUSTRALIA, November 29, 1930.

CORONARY OCCLUSION.

IT is generally agreed that coronary occlusion in most cases occurs at rest or during routine activities; however, in a small proportion such attacks closely follow trauma, unusual physical exertion or emotional stress. Louis A. Kapp[1] feels that this latter aspect of an all too common disease has been often neglected and the possibility of a causal relationship too lightly dismissed by those whose approach is statistical rather than clinical. With only the usual circumstances in mind, he thinks that some patients may not receive adequate treatment, their condition being called coronary insufficiency, and that others in industry may be deprived of a reasonable claim for compensation. The author presents a clinical study of 42 cases of coronary occlusion selected from 102 cases of acute "heart attack" closely following trauma or unusual physical exertion. Thus this study does not give any indication of the relative importance of antecedent trauma or exertion in coronary occlusion as a whole. In his report the term coronary thrombosis is sometimes used when the more general term coronary occlusion is intended and these terms are interchanged with myocardial infarction, which is sometimes confusing. In a review of the literature the author finds one group of observers who hold the view that physical effort or emotional stress may frequently be related to coronary insufficiency, but only rarely or coincidently to coronary occlusion. The work of A. M. Master, S. Dack and H. L. Jaffe[2] indicated that the onset of occlusion was related neither to the state of rest nor to the activity of the patient at the time. Kapp quotes other investigators who have found what he regards as a convincing causal relation between severe exertion and coronary occlusion and illustrates this with selected case histories. In addition, he argues, prolonged coronary insufficiency may itself lead to myocardial infarction, the result depending on the duration and degree of muscle ischæmia, and it is not possible to distinguish in all cases between coronary insufficiency and coronary occlusion with infarction, even when aided by serial electrocardiograms. Several mechanisms are suggested whereby the coronary vessels may become occluded following exertion. Subintimal and intramural hæmorrhages are more likely to occur with a rise in blood pressure during severe exertion; which may also cause a tear in the wall of an inelastic vessel with rupture of an atheromatous abscess, or dislodgement of a plaque. However, at the most this suggests that effort is only a minor precipitating factor in the occlusion of already diseased vessels, the state of the vessels being of much greater importance than the severity of exertion. In a recent discussion on heart disease in industry in the editorial columns of this journal[3] the evidence was found not to favour effort as a cause of coronary occlusion.

Louis A. Kapp's criteria—chiefly some unusual exertion followed promptly by cardiac symptoms—by which a causal relationship with trauma is determined superficially may seem reasonable. When analysed on this basis there was a definite relationship in 13 of his 42 cases; relationship was possible in six, doubtful in five and absent in 18 cases. The lack of any control series leaves him open to the criticism that the apparent relation he finds is mere coincidence. His argument is based on selected cases, a method which is often misleading, and until he produces a more soundly based analysis his conclusions are bound to be viewed with scepticism.

[1] *Ann. Int. Med.*, February, 1954.
[2] *J.A.M.A.*, 1937, 109: 546.
[3] M. J. AUSTRALIA, February 13, 1954.

Abstracts from Medical Literature.

PATHOLOGY.

Synovial Sarcoma of the Tibia.

J. DOUGLAS HICKS (*J. Path. & Bact.*, January, 1954) describes two cases of synovial sarcoma of the tibia. He states that the resemblance to the so-called adamantinoma of the tibia suggests that this tumour also is synovial in type, and examination of previous reports reveals much to support this hypothesis. The stroma is an integral part of the tumour. Squamous differentiation and the relation to basal-cell carcinoma are discussed. Reference is made to the clinical behaviour and to older reports of tumours of the tibia. The author states that the concept that adamantinoma of the tibia is a synovial tumour removes the need for those dubious assumptions which hitherto were used to explain its development.

Juvenile Naso-Pharyngeal Angiofibroma.

STEPHEN S. STERNBERG (*Cancer*, January, 1954) states that a pathological study of 25 cases of juvenile naso-pharyngeal angiofibroma has indicated that these tumours, despite a variable histological pattern, have an underlying basic vascular structure. This vascular make-up is evidence for the belief that this tumour represents a distinctive variant of the angioma, with a unique stroma and stromal cell. The occurrence of nearly all angiofibromata during the relatively limited age period of adolescence, the instances (though rare) of partial spontaneous regression or improvement as maturity is approached, and the fact of their occurrence only in males (in this series) indicate that these tumours are sex-linked and age-linked and probably influenced by some as yet unknown endocrine factors.

Congenital Pneumonia.

R. BARTER (*J. Path. & Bact.*, October, 1953) states that inflammatory lesions in the lungs were present in 19% of still-born fœtuses and live-born infants surviving for less than six hours, whose lungs were examined microscopically. It is highly probable that the lesions in the live-born infants commenced before birth. Any inflammatory lesion in such lungs constitutes congenital pneumonia, but two types of lesion are more or less characteristic of this condition: (*a*) a widespread exudation of leucocytes into the alveoli with round-cell infiltration of the interstitial tissues; (*b*) round-cell infiltration around bronchioles containing inflammatory exudate or obviously aspirated material. The latter is more common in the very premature fœtus or infant. Lesions similar to those seen in human subjects can be reproduced in fœtal rabbit lungs by injecting organisms into the amniotic sac. The infections produced experimentally are probably more virulent than those occurring spontaneously in the human subject before or during labour. The experimental observations suggest that the first inflammatory lesions to develop are in the bronchioles, and that exudation into the alveoli occurs later; but that in a few cases focal inflammatory infiltration due to aspirated material also occurs in the periphery of the lung. In the relatively highly virulent experimental infection of the amniotic sac, the fœtuses in most cases appear to develop septicæmia. Septicæmic lesions can be induced in the lungs of fœtal rabbits by the injection of organisms into the placenta and are almost entirely confined to the interstitial tissues. Aspiration of purulent material from the birth canal may be responsible for the dense cellular exudates seen in the bronchi and bronchioles in the human subject, but it is equally probable that such exudates may develop from inflammation in fœtal lungs. Congenital pneumonia of moderate extent is acceptable as a cause of intrauterine or early neonatal death, but usually there are also other complications of pregnancy or labour additional to rupture of the membranes over a prolonged period.

Hypertrophy and Hyperplasia of the Pancreatic Islets in the Newborn.

B. S. CARDELL (*J. Path. & Bact.*, October, 1953) states that in 38 infants of non-diabetic mothers the amount of islet tissue was found to range from 0·7% to 2·6% of the total pancreatic tissue, with a mean of 1·5% ± 0·45%. In 18 infants of diabetic mothers the amount of islet tissue ranged from 1·8% to 9·9% of the total pancreatic tissue, with a mean of 4·35% ± 2·27%. Thirteen showed an increase of islet tissue, which appeared to be due mainly to an increase in the β cells. There was a close relation between the amount of islet tissue and the infant's body weight. In three cases of *erythroblastosis fœtalis* the amount of islet tissue lay within the normal range. It is concluded that there is a wide normal range in the amount of pancreatic islet tissue in newborn infants, but that a true increase is common in the infants of diabetic mothers. It may also be found in the infants of "pre-diabetic" mothers and, less often, in subjects of *erythroblastosis fœtalis*. Its occurrence in other infants is rare. The increase of islet tissue in the infants of diabetic and "pre-diabetic" mothers is possibly due to overproduction of the maternal anterior-pituitary diabetogenic growth hormone.

Study of the Supportive Structures of the Uterus by Levator Myography.

B. BERGLAS AND I. C. RUBIN (*Surg., Gynec. & Obst.*, December, 1953) set out to determine the anatomical and functional relationship of the uterus to the *levator ani* muscle in the living subject, under normal conditions and in cases of prolapse. It is emphasized that in the cadaver, owing to the loss of tone of the muscular pelvic diaphragm, the anatomical relationship between the uterus and the pelvic floor is greatly altered. A new method has been presented for the study of the anatomy and physiology of the *levator ani* muscle in the living female by the use of X-ray myography. Under normal conditions with intact muscular pelvic diaphragm, the levator plate has an almost horizontal course, and the uterus is situated over this structure. With increased intraabdominal pressure, the uterus is forced against the levator plate, which provides it with adequate support. In the defective muscular pelvic diaphragm, whether due to birth injury or to other structural and functional impairment, the levator plate assumes an oblique or vertical position. This results in an altered relationship of the uterus to the levator plate and in an increase of the longitudinal diameter of the pelvic floor aperture. The increase of the transverse diameter of the hiatus is due to impairment of the levator crura. The disturbed anatomical relationship of the uterus to the levator plate is chiefly responsible for the loss of the support normally given to the uterus by the levator plate.

Sarcoma Botryoides in the Female Uro-Genital Tract.

WILLIAM B. OBER AND JOHN H. EDGCOMB (*Cancer*, January, 1954), in a study of ten cases of *sarcoma botryoides* in the female, present the major clinical and pathological features of the tumour. The tumour is defined as having origin from the mesenchyme of the uro-genital ridge and is a juvenile member of the series of mixed tumours of the uro-genital region. Historical concepts regarding this and related tumours have stressed the probability that the cellular background for the development of *sarcoma botryoides* is determined in embryonic life, albeit the initiating stimulus remains unknown and the precise cellular mechanism of malformation is debatable. The clinical behaviour of *sarcoma botryoides* varies within limits depending primarily upon its site of origin. In eight of the cases in this series death occurred within five years after treatment, or the patient had residual tumour when last interviewed; one patient survived for a year after cystectomy, but the outcome is not yet determined; one patient is living and free from evidence of recurrence two and a half years after operation. Search of the literature reveals only one case in which a five-year survival following operation has been achieved.

MORPHOLOGY.

Diencephalic Temperature Regulation.

W. R. MCCRUM (*J. Comp. Neurol.*, April, 1953) describes the results of a series of experiments performed in an attempt to establish the anatomical localization of temperature-regulating mechanisms in the hypothalamus and pre-optic area of the forebrain. In addition, the source of the heat in morphine hyperthermia was investigated. Observations on morphine response in the normal cat were compared with those on the response in cats with hypothalamic lesions, frontal lobectomies and thyroidectomies. The effect of curarization on the morphinized animal was also studied. Electrolytic lesions were stereotactically placed throughout the hypothalamus and pre-optic area in 22 cats. At the conclusion of the experiments the cats with lesions were sacrificed and the changes in behaviour correlated with the anatomical findings upon examination of the brain. The author states that from the results of the experiments it seems evident that a transient hyperthermia may appear post-operatively after lesions in or about the hypothalamus but not impinging upon areas known

to be concerned with temperature regulation. In such cases the hypothalamic thermoregulatory centres are only indirectly affected by the lesion; thus temperature alterations are transitory. On the other hand, destruction of a sufficient amount of the thermoregulatory centre produces permanent body temperature changes. There appears to be present in the hypothalamus a neural mechanism which prevents the heat-production centre, in response to specific stimulus, such as morphine, from producing a body temperature high enough to harm the animal. Bilateral lesions placed along the wall of the third ventricle in the hypothalamus may interfere with this regulation, and as a result body temperature in response to certain stimuli may exceed the normal limit without a concomitant depression of heat-loss activities. The heat-loss mechanisms and heat-production mechanism appear to be diffusely intermingled with one another throughout an area extending from the anterior tuberal region of the hypothalamus to the anterior commissure. Furthermore, the distribution of the component parts of the thermoregulatory mechanism may vary from animal to animal, so that consistent results from lesions of the same area in different animals cannot be obtained, or, conversely, post-operative temperatures may be either above normal or below with similarly placed lesions in different animals.

Intersemilunar Cerebellar Ridge of the Occipital Bone.

J. KRMPOTIC-BOHACEK (*J. Anat.*, October, 1953) gives an account of the shape, variations and significance of a bony ridge in the cerebellar fossa of the occipital bone. The ridge is not mentioned in anatomical texts, though it figures in many anatomical illustrations. The author examined 866 occipital bones and 221 cerebella with special regard to the features on the occipital surface of the hemispheres. He states that the ridge separates two shallow depressions, the upper lodging the superior and the lower lodging the inferior semilunar lobules of the cerebellum, and that it can be correlated with the horizontal fissure of the cerebellum. The ridge was present in 80% of cases and varied in its degree of development, being continuous (64% of cases), interrupted or only present in part.

Intrinsic Bone Form.

F. GAYNOR EVANS (*Am. J. Phys. Anthropol.*, Volume II, 1953) demonstrates that many of the techniques employed by engineers for stress-strain analysis in engineering structures and materials are also applicable for studying similar phenomena in bones. By these means one may study the stresses and strains produced in bones under controlled conditions of loading and orientation in which the magnitude of the load or the energy applied to the bone, as well as its point of application and direction, can be controlled. It is thus possible to obtain some idea of the behaviour of the bone as a mechanical structure or unit. It is, of course, obvious that the experimental conditions employed in such studies are far more simple than those occurring in the bones of a living animal. In the latter case, stresses and strains to which the bone is subjected are constantly changing during different phases of locomotion or movement. It has also recently been shown that a reduction in the blood supply to a bone has pronounced effects on its physical properties and hence its mechanical behaviour. Regional differences in the physical properties of the human femur have been studied by Evans and Lebow, who found that the areas subjected to tensile strain, as revealed by "Stresscoat" studies of the femur under static and dynamic vertical loading, also have the highest average tensile strength. More recently the same authors have shown considerable differences in the physical properties of the various bones of the inferior extremities in a single individual. All these factors, as well as age, sex, state of health, and nutrition, influence the biochemical behaviour of bones.

Origin of Colostrum Cells.

S. ENGEL (*J. Anat.*, October, 1953) states the conflicting views on the origin of the cellular elements of the colostrum. The opinion most commonly held is that large and small colostrum cells originate from mesenchyme, enter the colostrum through the epithelium of the mammary alveoli and ducts, and are therefore wandering cells. Others state that both wandering and epithelial cells may be present in the colostrum, while still others suggest that the majority of the cells are epithelial in origin. The present investigation deals especially with the histological changes in the human mamma (58 cases) in the early post-parturitional stage, and supports the view that the majority of colostrum cells are epithelial in origin. The alveoli of the non-lactating mammary gland are lined by a double row of cells. The basal layer is best recognized when the breast is under increased hormonal stimulation. The basal layer is composed of the secretory cells, whereas the internal layer protects the former and is cast off as soon as the breast functions. The cast-off cells of the internal layer appear as small colostrum cells. The large colostrum cells take their origin from proliferated and degenerated cells of the ducts. It is concluded that the majority of colostrum cells are epithelial in nature; a small minority belong to the family of wandering cells.

Organization of the Cerebral Cortex.

D. A. SHOLL (*J. Anat.*, October, 1953) states that the manner of transmission of the nerve impulse from one neuron to another in the cerebral cortex is unknown. Few terminal buttons are seen on the cell bodies of cortical neurons, and the nature of the synapsis is obscure. The author attempts to assess the general principles of neuronal interaction from a consideration of the geometrical relationships subsisting between the neurons and between neurons and the incoming axons. He measures the density of packing of the cells and the pattern of branching of the dendrites and axons. As examples of his findings, he estimated that in the visual area 25% to 30% of the cortex is occupied by perikarya (cell bodies or cytons), while the dendrites in this region occupied 20% to 30% of the cortical volume. The region infiltrated by the dendrites of one nerve cell or neuron contains between 2000 and 4000 perikarya. A connective zone and a connective field are defined for a neuron and it is shown that the density of the connective field falls off exponentially with distance from its centre. An analogy between the visual cortex and a statistical machine with a random input is considered.

Medial Root of Optic Tract.

E. WOLFF (*Brain*, September, 1953) states that the posterior part of the optic tract is generally described as being divided by a groove into lateral and medial root and that the medial root is not visual in function, but is the commissure of Gudden joining the two medial geniculate bodies and is an auditory commissure. The author submits evidence that there is no line of demarcation between the medial and lateral portion of the tract, and that there is no real difference in their termination in the lateral geniculate body. Both lateral and medial portions of the optic tract are therefore visual in function, and the medial is not Gudden's (auditory) commissure. Gudden demonstrated his commissure in the rabbit, but there is no evidence that it occurs in man.

Pyramidal Tract Fibres from Temporal and Occipital Lobes.

F. WALBERG AND A. BRODAL (*Brain*, September, 1953) made cortical lesions in the temporal and occipital lobes of adult cats, and the ensuing fibre degeneration has been studied in silver-impregnated sections (Glees's method). After such lesions have been made, degenerating fibres can be traced via the internal capsule, the cerebral peduncle, the longitudinal bundles of the pons and the medullary pyramid to the lumbar level of the spinal cord. In the cord degenerating fibres and collaterals enter the grey matter. Some of these cortico-spinal fibres pass via the *corpus callosum* to the contralateral internal capsule and descend in the contralateral cerebral peduncle and pyramid. In the pyramid decussation a considerable proportion of the fibres cross from the side of the lesion to the contralateral side, while some pass in the opposite direction. Most of the degenerating fibres in the cord are found in the lateral cortico-spinal tract on the side contralateral to the lesion, but also the homolateral lateral and both ventral cortico-spinal tracts contain degenerating fibres. The temporo-spinal and occipito-spinal fibres are of varying diameters. Some are thick, but most of them are medium-sized or thin. Two cats subjected to cortical lesions of the "motor" area were studied as controls. The ensuing cortico-spinal fibre degeneration was essentially as in the animals having lesions of the temporal and occipital lobes, but quantitatively much greater. Also some of these fibres cross in the *corpus callosum*, and degeneration can be traced in all four cortico-spinal tracts to the lumbar level. It is concluded that the temporo-spinal and occipito-spinal fibre systems demonstrated in the present study, as well as the ascending pyramidal fibres studied previously by the authors, must be considered as integrating components of the pyramidal tract. Reasons for this are given and some comments made on the possible functional role of these fibres. The *corpus callosum* cannot be considered a purely commissural structure.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

CIV.
THE TEETH OF CHILDREN.

THE general medical practitioner's interest in children's teeth may be discussed under four main headings: (*a*) The eruption of the deciduous dentition. (*b*) The deciduous dentition (sixth month to sixth year). (*c*) The "mixed" dentition (sixth to twelfth year). (*d*) The permanent dentition (after the twelfth year).

Eruption Period of the Deciduous Dentition.

The family physician is often consulted about cases of infantile "teething troubles". While it is true that delayed or difficult eruption of the deciduous teeth may cause serious systemic reaction, the majority of infants' teeth erupt with no more than local soreness and general irritability lasting a day or two. Many lower incisors make their appearance in the mouth without previous sign or symptom; others are helped to erupt by allowing the infant to bite on a rusk or a clean bone ring. If, in the rare case, the gum over the erupting tooth remains red and turgid for a period of days and the child is seriously upset, it may be of help to perform incision. This is best done by having the nurse wrap the child in a blanket and hold him on his side over her knees. The doctor's or nurse's little finger can be used as a gag to keep the mouth open while a crucial incision is made down to the crown of the tooth. The patient is then turned face downwards until the slight bleeding has stopped. Sometimes incision is insufficient to alleviate the condition, and at a second operation a small section of the gum tissue must be dissected away over the crown of the tooth. The area should be swabbed with a mild antiseptic lotion, such as boroglyceride, before and after operation.

Unusually, a child may be born with one or more deciduous incisors erupted, and these are seen almost invariably in the lower jaw. On other occasions such teeth may erupt prematurely. The question put to the family physician is "Should these teeth be removed or left alone?". Unless they are obviously malposed or loose, or unless they cause trauma to the mother's nipple during nursing, they can usually be left alone with safety. If it is necessary to remove them, they can be extracted without any form of anæsthesia as the infant's threshold for dental pain is high. The technique for handling the patient during the extraction is the same as described for gum incision.

The Deciduous Dentition (Sixth Month to Sixth Year).

On the average, all the teeth of the deciduous dentition are erupted and in occlusion by the twenty-fourth month, and it is not too early for a dentist to inspect the mouth at this age. The well baby should go to the dentist just as he goes to the pædiatrician. Regular six-monthly dental visits should be arranged from this time onwards, and the "dental home care habit" should be commenced. Every night before the child is put to bed the parent should wipe the crowns of all the teeth with a soft cloth rinsed in warm water to which a small quantity of a palatable toothpaste may be added. After this wiping the mouth should be gently rinsed with warm water. As the child gets older he should be encouraged to take over the tooth cleansing, although the parent must constantly supervise. As soon as possible the toothbrush must be used—first by the parent and later by the child. The appropriate type of brush is indicated by the dentist, who also teaches the child how to use it. Preferably the right side of the mouth should be cleaned with the brush held in the right hand, and *vice versa*. It is important to train the child always to use the looking-glass over the hand-basin while cleaning the teeth. Objections to this usually can be overcome by pointing out that the hands are never washed without being looked at, and that the teeth deserve at least equal consideration. Incidentally, appropriate fittings must be made in the bathroom so that the child can look in the mirror during tooth cleansing.

The general medical practitioner is in an advantageous position to give early encouragement to sound oral health habits. Also, he can help to prepare the child psychologically for regular dental treatment. This can be done by positive suggestion on his part, and by warning the parents about foolish remarks in front of the child. Some parents imply by their conversation that a visit to a dentist is something to fear. A mother, within the hearing of her child, may tell a friend that she is taking the child to the dentist, and that she hopes "the dentist will not hurt"; or she will discuss her own dental experiences and exaggerate the pain she has suffered. The child not infrequently reacts unfavourably to such comments, and becomes intractable on the day of the dental appointment.

With the proper encouragement from the family physician and from the parents, the young patient will meet the dentist without real fear, and thus will be saved much mental distress and unnecessary pain in the years to come.

During the period of the deciduous dentition it is also necessary to inculcate correct dietary habits and discipline which will carry the child through school age and adolescence. Teaching must be by precept and example on the part of the parents. Unless parents demonstrate that they themselves take dental home care and correct dietary habits seriously, it is useless to expect a child to pay more than a token allegiance to these matters.

Dental caries is a disease of "civilization" and is the most prevalent of all disease processes affecting children. Whatever other causes may or may not come into action, it is known that excess of refined carbohydrate in the form of sugar and white flour products in the diet is associated with an increased incidence of dental caries; conversely a restricted intake of sugars and refined carbohydrates produces a proportional decrease in caries (Lilienthal *et alii*, 1953).

Young children can be brought up to take whatever limited amounts of sweets, cakes, pastries and sweetened beverages are allowed, as a ration with their meals, and not between them. Between-meal snacks, if allowed, should consist of something tough and fibrous, such as an apple. The pernicious habit of bribing a child to go to sleep by giving him a biscuit to eat in bed is only mentioned to be condemned. While it is most desirable for teeth to be cleaned with a toothbrush immediately after every meal, this is often impracticable when the child eats away from home. However, the use of the "mouth rinse" after food is always possible. A mouthful of water is taken and energetically sucked in and out between the teeth, and the tongue is moved freely over the outer, inner and occlusal surfaces of the crowns. The water is then spat out and the process repeated twice. By this means a great deal of lodgeable carbohydrate debris is voided with a consequent lessened risk of caries.

If carious cavities unfortunately develop in deciduous teeth, they should be filled if it is at all possible to do so. A full complement of deciduous teeth is necessary for proper mastication of the gradually changing diet of young childhood; also it is necessary to maintain the stability of the dental arches while bone growth in the face and jaws is progressing. A vital period of facial and maxillary growth extends from the third until the end of the seventh year. If a deciduous tooth is to be filled, the type of filling to be inserted must be left to the judgement of the dentist, but it is stressed that, in general, as high a standard of conservation technique is required as for a permanent tooth.

Premature loss of deciduous teeth is one of the well-known causes of malocclusion in the permanent dentition. When a deciduous molar is lost prematurely through caries, the dentist must determine whether or not a "space retainer" is required to prevent drift among the erupting permanent teeth. The use of this simple appliance can prevent many months of orthodontic treatment in the future, and can save the parents much expense.

The Mixed Dentition (Sixth to Twelfth Year).

In the period from the sixth to the twelfth year both deciduous and permanent teeth are present in the mouth.

The first permanent molar takes its place in the arch immediately behind the second deciduous molar during the sixth year. It is usually the first of the permanent dentition to erupt; and as it is has no predecessor, it is often mistaken by parents for a deciduous tooth and unfortunately neglected. Its importance is such that it is known as the "keystone of the dental arch" because it maintains the jaws in correct relationship during the ensuing period of rapid bone-growth. Every effort should be made to conserve it, because its loss during early childhood almost inevitably leads to some form of malocclusion.

Supernumerary teeth are occasionally found during the mixed dentition period; they may be erupted or non-erupted. The latter are demonstrated by X-ray films taken for routine examination or for orthodontic requirements. These teeth are usually found in the pre-maxillary region, and they must be removed at a convenient early opportunity; if left *in situ* they may cause maleruption of contiguous teeth, or they may become infected, with consequent local osteitis.

It is not uncommon to find some form of congenital hypoplasia of the enamel in the crowns of the permanent incisor teeth as the result of a systemic disturbance when calcification was occurring in the tooth germs. For example, an attack of one of the exanthematous fevers between the ages of one and two years can produce a "chalky" appearance of the incisal portion of the crowns of the upper or lower central incisors.

Other causes of hypoplasia of the enamel in children include (i) serious systemic disease in infancy, (ii) malnutrition in infancy, (iii) consumption of drinking water with a fluoride content greater than one part per million, (iv) congenital syphilis, and (v) congenital ectodermal dysplasia.

The period of the mixed dentition is one of the most hazardous as regards the increase of dental caries. This disease is no respector of age or income groups, and those children whose parents can afford the best of food and the best of dental treatment do not necessarily have the best teeth.

> The family physician notices dental caries in children often before a dentist is consulted; he is frequently asked for advice on what to do to prevent caries, and usually struggles with this insidious ailment in his own family. The fight is, as a rule, frustrating, and the teeth of the physicians' and dentists' children are not better than those of any average group in a community. Consequently, the medical practitioner looks with some confusion and hope to the reports about progress in the dental field in the prevention of caries. (Neumann, 1951.)

As previously mentioned, the incidence of dental caries appears to increase proportionately with the increase of sugars and refined carbohydrate products in the diet. These foodstuffs would appear to be especially deleterious in civilized communities when they are taken (*a*) in excessive amounts, (*b*) in sweetened beverages, especially proprietary "soft drinks", (*c*) in a sticky or "tacky" or "lodgeable" form, such as soft pastries, cakes and "toffee-like" sweets, and (*d*) in between meals, especially before retiring at night.

Approximate figures for the annual consumption of sugar *per capita* are 132 pounds in Australia, 120 pounds in the United States of America, 30 pounds in Spain, and 26 pounds in Italy.

During World War II, when the sugar ration was drastically reduced, and bread made from white flour was practically unprocurable in both Great Britain and Norway, the teeth of the children in both countries showed a striking decrease in dental decay. However, during peace it is not possible to control a democratic nation's diet, and the best that can be hoped for at present is a workable compromise between regular dental treatment, home dental care and a "realistic modification" of diet, together with increased research into preventive measures.

Mass therapeutic preventive measures have been favoured in parts of the United States in recent years, and much has been written about the value of fluorides as caries-inhibiting agents, used both by topical application and in the drinking water.

The results of topical application of fluoride solutions to the crowns of the teeth appear to be varied, limited in duration and at times of no significant clinical value (Darling, 1952). The technique of application requires considerable care and attention to detail. If topical application of a fluoride solution is decided upon, it should be carried out at times which would allow optimal effects on newly erupted teeth—namely, at the ages of three years, seven years and twelve to thirteen years.

Over the past six or seven years, fluoridation of communal water supplies in many centres in the United States has produced a significant reduction in the caries incidence in young children. Some investigations in England indicate that this beneficial effect decreases after adolescence. The possibility of long range general and/or local deleterious effects of fluorine introduced into a water supply cannot be ignored. However, until such effects are proved—and up to date they have been neither proved nor disproved—artificial fluoridation of drinking water can be regarded as a valuable measure in the partial prevention of caries.

> While introduction of fluorine should be welcome for want of anything more effective, it is not justified to tack high hopes on it. Even though it is effective in reducing caries, its deficiency is certainly not the cause of decay, as some areas with very little fluorine in the water have been found to be relatively free from caries. (Neumann, 1951.)

Hence, fluoridation at best is only part of the answer to caries prevention and control; regular dental treatment, reasonable control of diet and home dental care remain more important than ever.

Permanent Dentition (After the Twelfth Year).

After the twelfth year is a period when accidental injury to the permanent upper incisors is not infrequent. The child is often taken first to the family physician because of concomitant injury to the upper lip. It is important that the case be referred to the dentist at the very earliest opportunity in order that dental conservation measures may be instituted without loss of time.

The cuspids (canines) are the last deciduous teeth to be shed, and they are usually replaced by their permanent successors by the end of the twelfth year. If the latter are not present by the end of the thirteenth year, X-ray films should be taken to make sure that there is no obstruction to eruption. Systemic and local factors may delay normal eruption; and when local factors positively prevent eruption the tooth is said to be "impacted". It may be out of its normal position or deflected in its axial alignment, or its crown may be locked against the root of another tooth. A mild degree of impaction may right itself over a period of twelve to eighteen months, and periodic check X-ray films can reassure the parents. In a more serious case, specialist advice should be obtained, and one of several methods of treatment is usually possible; these include (*a*) removal of the unerupted tooth—a delicate operation if the contiguous teeth are to be preserved; (*b*) surgical exposure of the crown and removal of bony impediment to eruption (the mucoperiosteal flap is repositioned at the end of operation); (*c*) permanent exposure of the crown and insertion into it of a stainless steel pin, to which a wire can be attached for orthodontic traction.

From the age of twelve years onwards, a child faces great changes in height and weight, together with social, physiological and educational transition. The summation effect of all these tends to make him or her forget good health habits of previous years, including those of oral hygiene. As most of the teeth of the permanent dentition are erupted and in use, it is vital that every encouragement should be given to regular dental care, especially as another dental disease can make its appearance at this age, namely *pyorrhœa gingivæ*. In an uncared-for mouth of a pre-adolescent, chronic or subacute gingivitis is frequently present; and if the condition is allowed to progress, ulceration in the gingival trough can occur with the establishment of true pyorrhœa. There is a greater incidence of pyorrhœa among older children than is generally appreciated. The disease is painless, and this quality is one of its greatest dangers because it can insidiously exert a serious cumulative effect on the general health of the patient.

A. B. P. AMIES,
Melbourne.

References.

DARLING, A. I. (1952), "Caries: The Present Position", *Brit. Dent. J.*, 93: 279.

LILIENTHAL, B., GOLDSWORTHY, N. E., SULLIVAN, H. R., and CAMERON, D. A. (1953), "The Biology of the Children of Hopewood House, Bowral, New South Wales: I. Observations on Dental Caries Extending Over Five Years (1947 to 1952)", *M. J. Australia*, 1: 878.

NEUMANN, H. H. (1951), "Medical Progress and Dental Decay", *New York J. Med.*, 51: 1749.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

A STRANGE ACCIDENT.[1]

[From White's "Journal of a Voyage to New South Wales".]

15 May, 1787.

AN accident of a singular nature happened today. Corporal Baker of the Marines on laying a loaded musquet down, which he had just taken out of the arms chest was wounded by it in the inner ankle of the right foot. The bones, after being a good deal shattered, turned the ball, which, taking another direction had still force enough left to go through a harness-cask full of beef, at some distance and after that

[1] From the original in the Mitchell Library, Sydney.

to kill two geese that were on the other side of it. Extraordinary as this incident may appear it is no less true. The corporal being a young man and in good habit of body, I had the pleasure, contrary to the general expectation, of seeing him return to duty in three months, with the perfect use of the wounded joint.

Correspondence.

THE TRAINING OF MEDICAL STUDENTS.

SIR: Much has been recently written about the medical education of the University of Sydney, mostly in the lay Press. It has been implied that, as a result of lack of funds and overcrowding, the standards of teaching have been lowered. Whilst one cannot deny that we should never be satisfied with present results—for satisfaction means stasis, stagnation and retrogression—let us examine the present position of our medical education dispassionately.

At the University of Sydney there is no restriction on the number of students entering any faculty. The authorities responsible for this should be congratulated, for it appears to the writer that all who can reach a sufficient standard to matriculate into the University should be allowed to do so. As far as those who enter the medical course are concerned, the misfits, if any, automatically disqualify themselves as the course advances. This is a fairer way of exclusion than the various methods of selection of students.

Naturally this policy of unrestricted entry may lead to temporary overcrowding, as occurred recently in the post-war years with their huge influx of ex-service men and women, and at this point one must pay tribute to the responsible heads of all departments for the extraordinarily efficient way in which the numerous problems caused by this influx of students were solved.

There was no lowering of educational standards, because it is just as easy to lecture to four hundred as to two hundred students, even if the lecture hall is constructed of galvanized iron or is a little cold and draughty in winter. This does not apply to hospital rounds, where large groups of students cannot be instructed efficiently, so to supply the increased number of students with adequate clinical material, other hospitals with their staffs reinforced the already existing teaching hospitals, and at least one has been raised permanently to this latter status. Their results were at least as good as those of the older teaching hospitals, so that the standard of clinical instruction never depreciated.

In this way the ratio of teaching beds per student was kept at a reasonable level. To say that forty years ago there were ten teaching beds per student, whereas now there are only four, gives a false impression of the true position, because the rapid turnover of patients today, as compared with those days, allows the student far more clinical material than those figures imply.

Nowadays, too, teaching is more efficient and concentrated than heretofore. Each teaching hospital has a dean of its clinical school (or equivalent senior officer) who is helped by one or more student supervisors. Teaching is performed by the visiting honorary medical staff on the one hand, and the resident registrar staff on the other, both of whom have their own important functions to perform at different levels of teaching. The teachers, themselves, now are under greater discipline than previously. One can remember times when a group of students would wait in vain for hours for an honorary who did not appear. Now, if a teacher cannot come, his registrar takes his class. If this occurs too often, however, the honorary, no matter how senior, is asked to explain his dereliction of duty.

The whole outlook and aim of undergraduate medical education have changed with the years, a change that has occurred all over the world. The University now aims to produce doctors with a sound general medical education (necessarily mostly theoretical), not fully-fledged surgeons, physicians, research workers or even general practitioners. The diplomas (M.B. and B.S.) represent only the completion of this phase of instruction and merely entitle the recent graduate to become a hospital resident, where he can learn the general practical side of the profession. Once this is passed, then the doctor can go into general practice, preferably under the tutelage of a more senior man (and this is the only way to learn general practice, not as an undergraduate), or commence to learn a specialty under the ægis of one or other of the Royal Colleges.

This new orientation of outlook certainly implies no lowering of standards, but rather, in the long run, of considerably raising them. This can be shown by watching the careers of our recent graduates in countries abroad.

No matter how well financed any system of medical education is, it is concerned basically with three people, the patient, the student and the teacher, and its successful outcome will depend, therefore, on a good supply of clinical material, a good teacher who must be interested in teaching (and many research men have not this interest or flair for teaching), and lastly on the good calibre of the student. These alone are the important factors, and we have all of them here. Expensive and even extravagant visual and other aids to teaching are purely ancillary, and cannot replace good teaching, or make up for bad teaching, or make good doctors of poor students.

In the same way, if more money is made available to attract more good teachers, or to pay better those already here, or to build new or modernize existing hospitals, let it be remembered that the reputation of these hospitals depends not so much on the hospital architecture as on the calibre and ability of their staffs.

As far as the increasing cost of medical education is concerned, some of our philanthropists would be wise to give money, not for some specific purpose such as cancer or other dramatic form of research, but for the benefit of medical education as a whole and with no strings attached. Then, apart from helping more good teachers to train more good doctors to diagnose disease processes earlier, and so allow them to be treated earlier, every now and then there will arise a Florey, or a Gregg, or a Burnet, and the money will have been well spent. Wonderful laboratories with limitless money, while helping the man who has a genius for research, do not yield the secrets of Nature to inferiors no matter how well paid.

To sum up, then, it is maintained that the University has upheld its high standards of medical education in a changing world under at times admittedly adverse circumstances which have been and are being constantly overcome.

This letter is written because many lay and even medical people are alarmed by the statements of alleged lowering of medical teaching standards recently loosely made in the lay Press. Engaged in the clinical training of medical students, I have watched the trends of medical education for many years, both here and abroad, and Sydney compares well with the best.

Yours, etc.,

141 Macquarie Street,
Sydney,
June 19, 1954.

THOMAS F. ROSE.

SIR: The training of medical students is being discussed both in lay and medical journals, and the writers evidently suspect that the students are not being trained adequately. As a country practitioner it is only of recent years that I have worked with newly qualified men. Thus it is a shock to find that they know nothing of the everyday work of a general practitioner. They have learnt far more than we did forty years ago of blood chemistry and advanced medicine, but they do not know how to use the simplest instruments, or how to carry out everyday investigations. For instance, after six years of study they have had no opportunity of learning how to insert or remove sutures, have never handled a hypodermic syringe or an ethyl chloride bottle. They do not know how to apply simple dressings or adjust slings, nor have they ever given a dose of penicillin. They have not witnessed common operations such as appendicectomies and tonsillectomies, nor have they had any instruction whatsoever in simple instruments. They have not been taught how to inspect a child's ear drum, clean a discharging ear or take a child's temperature in the rectum.

In fact, they have learnt nothing of the everyday work of a general practitioner. Yet these graduates can rush off and do locums or sail away as ship's surgeons. True, they are supposed to be trained in practical procedures during their resident years, but in large hospitals they may not have the opportunity of working in the casualty department or children's wards, and in other hospitals they may not meet honoraries who are able or willing to train them in the elements of their work.

Another matter that I would mention is that recent graduates tend to regard the patients as cases, rather than as individuals, and have little appreciation of the fact that people's minds are as important as their bodies.

I think that it is time that those who frame the medical curriculum realize that these young people need to be trained in the work that they will certainly have to do, rather than spend most of their time acquiring much theoretical knowledge about diseases they may never encounter.

Yours, etc.,

Armidale, New South Wales, June 22, 1954.

ELLEN M. KENT HUGHES.

TREATMENT OF THE COMMON WART BY X-RAY THERAPY.

SIR: It is valuable to know that Dr. Belisario has noted the occurrence of telangiectasia on the fingers and other areas of thin skin, long after the radiation treatment of warts. Younger practitioners can only learn such facts from the wisdom of the experienced, and I am persuaded to restrict radiotherapy, except in special cases, to warts on the soles and heels. As I pointed out, it is these sites, however, which demand effective treatment.

Careful study of the text will reveal no anomaly in the statements regarding radiation technique. By inserting the word "in", Dr. Belisario would try to suggest the total time was three weeks, but the text does not suggest this, and the following line refers to fourteen days to remove any doubt. The cure rate quoted is quite genuine. This is a small city, and we have many opportunities to know the fate of our patients. The careful follow-up was referred to. Most of these patients continued to return for observation, without further fees, until I was satisfied.

This was the point of the article; not to advocate any particular radiotherapeutic technique. It is obvious that any technique, however good, will result in failures, if applied in a routine way. Neither surgeon nor physician pins his faith to a standard treatment. The patient presents his own particular problem. We must keep this in mind even when we have at hand a reliable physical modality. The article illustrates what can be obtained from a method when it is used with perseverance, patient observation and with continual regard to basic medical doctrine.

It is true that X-ray therapy routines when used for cancer can only be assessed at a five to fifteen years' time interval; but surely this is a different problem, and complete satisfaction will result if we get rid of the wart.

Yours, etc.,

Perth, June 20, 1954.

ALAN J. M. NELSON.

THE MANAGEMENT OF CHRONIC HYPOPARATHYROID TETANY.

SIR: The article in your journal of June 19 by Dr. C. R. B. Blackburn on "The Management of Chronic Hyoparathyroid Tetany" prompts me to ask why no provision is made in the list of "general pharmaceutical benefits" for the treatment of these unfortunate individuals? Without treatment they are subject to a host of major complications, even endangering life. Treatment when begun must be life-long and thus very costly. It therefore seems to me that the inclusion of calcium and "Olvita D" (or similar preparation) in the list of benefits is an urgent necessity. I have previously made this suggestion to the Department of Health, but without effect. Perhaps this note will catch the eye of someone with more influence than I possess.

Yours, etc.,

Warrnambool, Victoria, June 23, 1954.

J. K. GARDNER.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JUNE 19, 1954.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	2	4(4)	2	..	..	..	..	..	8
Amœbiasis	..	..	..	..	2(2)	..	..	..	2
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	2	..	..	..	2
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	2(2)	..	..	..	..	..	..	2
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	15(15)	3(3)	..	..	..	1	..	19
Diphtheria	24(17)	11(3)	4(2)	..	2(2)	..	1	..	42
Dysentery (Bacillary)	..	..	1	..	..	..	..	..	1
Encephalitis	1	1(1)	..	2(2)	..	..	..	..	4
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	21(7)	11(4)	..	..	4(2)	..	..	..	36
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	2(1)	..	..	..	..	..	2
Malaria	..	2(2)	..	..	2(2)	..	..	..	4
Meningococcal Infection	1	4	1	1	1	..	..	1	9
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	1(1)	..	..	..	..	..	..	..	1
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	7(2)	13(7)	5(2)	2(2)	6(2)	..	..	..	33
Puerperal Fever	1	..	..	1	..	..	..	..	2
Rubella	..	9(5)	..	..	3(2)	..	..	1	13
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	10(7)	15(9)	3(3)	2(1)	1	..	..	..	31
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	2	..	..	..	..	..	2
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	79(71)	19(12)	7(5)	1(1)	18(9)	..	..	1	125
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	4	..	1	..	..	..	5
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Public Health.

NATIONAL HEALTH ACT, 1953: PENSIONER MEDICAL SERVICE.

THE following three announcements were published in the *Commonwealth of Australia Gazette*, Number 38, of June 24, 1954.

REPRIMAND OF MEDICAL PRACTITIONER.

I, Earle Page, the Minister of State for Health, hereby give notice, in pursuance of sub-section (1.) of section 36 of the *National Health Act*, 1953, that I have this day reprimanded Cyril Edward Powell, of Caboolture, medical practitioner, following investigation and report by the Medical Services Committee of Inquiry for the State of Queensland established under section 110 of the Act concerning the conduct of the aforesaid medical practitioner in relation to his provision of medical services for pensioners and their dependants under Part IV. of the Act or under an arrangement made by the Director-General of Health under section 7 of the *National Health Service Act*, 1948-1949, and the National Health (Medical Services to Pensioners) Regulations.

Dated this fourth day of June, 1954.

EARLE PAGE,
Minister of State for Health.

REPRIMAND OF MEDICAL PRACTITIONER.

I, Earle Page, the Minister of State for Health, hereby give notice, in pursuance of sub-section (1.) of section 36 of the *National Health Act*, 1953, that I have this day reprimanded Maksymiljan Brenner, of 465 Oxford Street, Paddington, medical practitioner, following investigation and report by the New South Wales States Committee of Inquiry established under the National Health (Pensioners' Medical Services Committees of Inquiry) Regulations concerning the conduct of the aforesaid medical practitioner in relation to the provision of medical services to pensioners and their dependants under an arrangement made by the Director-General of Health under section 7 of the *National Health Service Act*, 1948-1949, and the National Health (Medical Services to Pensioners) Regulations.

Dated this thirty-first day of May, 1954.

EARLE PAGE,
Minister of State for Health.

REPRIMAND OF MEDICAL PRACTITIONER.

I, Earle Page, the Minister of State for Health, hereby give notice, in pursuance of sub-section (1.) of section 36 of the *National Health Act*, 1953, that I have this day reprimanded Reginald Murray Dunstone, of 175 Macquarie Street, Sydney, medical practitioner, following investigation and report by the New South Wales State Committee of Inquiry established under the National Health (Pensioners' Medical Services Committees of Inquiry) Regulations concerning the conduct of the aforesaid medical practitioner in relation to the provision of medical services to pensioners and their dependants under an arrangement made by the Director-General of Health under section 7 of the *National Health Service Act*, 1948-1949, and the National Health (Medical Services to Pensioners) Regulations.

Dated this thirty-first day of May, 1954.

EARLE PAGE,
Minister of State for Health.

Australian Medical Board Proceedings.

TASMANIA.

THE following have been registered, pursuant to the provisions of the *Medical Act*, 1918, as duly qualified medical practitioners: Davies, John Charles Allan, M.R.C.S., L.R.C.P. (London), 1943; Syme, James Robert, M.B., B.S., 1951 (Univ. Queensland); Rankin, John Duncan, M.B., B.S., 1954 (Univ. Melbourne); Gorman, John Michael, M.B., B.S., 1952 (Univ. Melbourne); Hancock, John Owen, M.B., B.S., 1952 (Univ. Adelaide).

The following additional qualification has been registered: Oakes, Jean Reid, M.F.A. (R.A.C.S.).

QUEENSLAND.

THE following have been registered, pursuant to the provisions of *The Medical Acts*, 1939-1948, as duly qualified medical practitioners: O'Connor, Arthur Gerard, M.B., B.S., 1953 (Univ. Sydney); Hensley, William Joseph, M.B., B.S., 1950 (Univ. Sydney); Coorey, Kevin Dominic Francis, M.B., B.S., 1952 (Univ. Sydney); Tudehope, James Robert, M.B., B.S., 1953 (Univ. Sydney); Smith, Ewart Austral, M.B., B.S., 1951 (Univ. Adelaide).

Deaths.

THE following deaths have been announced:

GERSTMAN.—Samuel Rudolph Gerstman, on June 22, 1954, at Toorak, Victoria.

DENHAM.—Harold Knight Denham, on June 15, 1954, at Brisbane.

MURRAY.—Gerald Aubrey Murray, on June 23, 1954, at Perth, Western Australia.

Diary for the Month.

JULY 13.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JULY 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JULY 19.—Victorian Branch, B.M.A.: Finance Subcommittee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Tasmania: Part-time specialist appointments for the north-west coast of Tasmania.

Editorial Notices.

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